LOW BACK Disorders Evidence-Based Prevention and Rehabilitation Stuart McGill 1LO\,\V]B3AC](( ]D)] §�)]RlD)]E]R§ Get the latest information on evidence-based prevention and rehabilitation approaches from an internationally recognized expert and learn how to apply the principles in industry, sport, and everyday settings . . Low Back Disorders: Evidence-Based Prevention and Rehabilitation provides professionals with the foundation to make the best clinical decisions for building the best prevention and rehabilitation programs. The author's unique research results quantify forces that specific movements and exercises impose on the low back. The text identifies commonly prescribed practices that are, in fact, contraindicated in promoting back health. Additionally, it provides appropriate strategies to offset injuries and restore function. Low Back Disorders will build your understanding of the science behind the practices so that you can use them effectively with clients and patients. The book presents a clear exposition of back anatomy and biomechanics. It also provides the evidence to dispel myths regarding spine stabilization exercise and various prevention approaches. For example, the quantification of stability will show which muscles are important and which exercises groove motor patterns to ensure spine stability. With Low Back Disorders, you will • gain valuable information on measured loading of the back during specific activities and apply it to avoid common-but counterproductive-practices in back rehab; • learn how to analyze patients' and clients' unique physical characteristics and lifestyle factors and tailor treatments and preventive measures to their individual needs; • learn how to help patients and clients achieve the low back stability required to prevent future damage; and • acquire the information necessary to prescribe effective exercises clearly and easily. Low Back Disorders is well organized and easy to understand. Part I provides a solid foundation on how the back works and how the back becomes injured. Part II shows you how to use this foundation to build evidence-based prevention programs by assessi�g risks, creating ergonomic interventions, and training personnel. Part III focuses on improving rehabilitation techniques and, specifically, quantification of spine stability to determine which exercises are important for spine stability for each person. ISBN 0-7360-4241-5 90000 Human Kinetics 9 7 Low Back Disorders Evidence-Based Prevention and Rehabilitation Stuart McGill, PhD University of Waterloo Canada Human Kinetics Library of Congress Cataloging-in-Public • .Ilion Data McGill. Stuart. 1957- Low hack disorders : evidence-ba�ed prevention and rehabilitation I Sttlan McGill. p. : cm. Include .. bibliographical reference .. and index. ISBN 0-7360-4241-5 (hard cover) 1. Backache. 2. Evidence-based medicine. IDNLM: I. Back Injurics--prevcllt ion&control. 2. Back hljuric .. --rchabilitation. 3. Evidence-Based Medicine. 4. Low Back Pain--prevcllion & control. 5. Low Back Pain--rehabilitation. WE 755 M4778 20021 I. Title. RD77I.B217 M42 2002 617.5'64052--dc21 2002005695 ISBN: 0-7360-4241-5 Copyright © 2002 by SlUan McGill All right ... re ... erved. Except for u�e in a review. the reproduction or utililation of this work in any form or by any elcctronic. mcchanical. or othcr mcan ... now known or hereafter invented. including xerography. photocopying. and recording. and in any information �lorage and retrieval system. b forbidden withollt the wrillcn pcnni ..... ion of tbe pllbli-,hcr. ACCluisitions Editor: Loarn D. Robcn!oJon. PhD: Developmental Editor: Elaine H. Mu .. win: Assistant Editor: Maggie Schwartcntraub: COI)ycdilor: Patsy Fonney; J)roofrcader: Erin Cler: Indexer: Cmig Brown: l)ermissiol1 Manager: Oalene Reeder: Graphic Designer: Fred Starbird: Graphic Artist: Yvonne Grirrith: Photo Manager: Lc�lic A. Woodrum; Cover Designer: Jack W. Davis: Cover Image: Primal Pictures; Photographer (interior): i..c,lic A. Woodrum. except where noted in Photo Credits (p.tge 280) or on individual photos: Art MalUlger: Kelly H�ndrcn: IIlustnltor (Mac): Tom Roberts: Illustrator (medical): Argosy: I)rinter: Sheridan Books Printed in Lhc United States of America 10 9 8 7 6 5 4 3 2 Iluman

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exacerbate existing ones. Other more specialized books promote spine stabilization exercises without quantifying stability, thus basing the recommended exercises on a good guess-albeit a guess based on years of clinical observation. A scientific foundation to justify the recommendations has been thin at best. This book is not about perpetuating clinical myth—it is about challenging them and proposing valid and justifiable alternatives. How the Book Is Organized The intention of this book is to provide the best available scientific evidence to optimize injury prevention and rehabilitation efforts. It is designed to assist those who work to prevent low back troubles and those who are charged with the responsibility of rehabilitating them. Part I begins with a scan of the landscape related to the low back question. Here the main issues will be introduced together with a brief introduction to the legislative and institutional constraints-some of which assist efforts to enhance back health while others act as direct barriers. This is followed with evidence as to how the low back structure and associated tissues work during conditions of good health and how they become injured. These chapters describe • how the spine works, • injury mechanisms involving individual tissues, and • injury mechanisms involving the full lumbar mechanism. Specific information-such as tissue loads, muscle activation levels, and measures of stability-is provided so that readers can meaningfully compare approaches when deciding how to prevent or rehabilitate low back injury. Thus, the first part provides the scientific foundation for the rest of the book. Identifying and implementing the best programs result from considering the evidence. Thus, part II supports evidence-based practice by applying the scientific foundation in part I to the prevention of low back injlly. Part III applies this same information to the rehabilitation of low back injury. xiii xiv Low Back Disorders Focus and Features of the Text This text will be of value for those involved in designing and delivering back trouble prevention and rehabilitation programs-physical therapists, physiatrists, chiropractors, kinesiologists, ergonomists, and olthopedic specialists. Low Back Disorders features a number of elements besides the basic text to ensure that all segments of the very diverse readership that deals with low back issues will be able to absorb and use the material. Each chapter introduction includes learning objectives. Special elements in some chapters highlight issues of methodological concern, for example, or simply provide further elucidation of some concepts or terms that are not common across disciplines. EvelY section that has clinical relevance contains highlighted statements identified as such. In addition, a wide variety of occupational and athletic examples throughout the book can be generalized to all activities of daily living. A major theme of this book is the benefits of blending rehabilitation efforts with ergonomics and prevention. Traditionally, ergonomists and industrial engineers have been responsible for prevention efforts mainly in the workplace, while the medical community has been responsible for rehabilitation. This natural division has caused an unfortunate compromise. Rehabilitation efforts will not be effective without preventing the cause of the tissue irritants, and prevention efforts will be retarded without an understanding of the variables obtained from medical investigations into the causes of tissue damage. No clinician will be effective if the cause of the patient's troubles is not removed. The expertise required for optimal prevention and rehabilitation can be found in the fields of ergonomics and rehabilitation medicine, which are naturally symbiotic. Many in the sport and business world have debated whether it is better to develop a stronger offense or defense. The obvious answer is that it is best to have both. I hope that this book will foster a better process resulting in more effective injury prevention and successful rehabilitation. Some Encouraging Words Some readers may be tempted to apply the material in palts II and III without digesting the foundation material contained in part I. This is a mistake. People who have attended my courses and were previously familiar with our published scientific papers

often state that they were unable to fully syntheSize the story and integrate the critical information. Once aware of the full story contained in the course and told in this book, they were much more effective at preventing low back disorders and prescribing exercises that help patients become better rather than worse. A Note to Lay Readers Although this book was written for clinicians, many lay people have bought copies from its initial print run. As a result, they have written me letters expressing their appreciation for their better understanding the science and their own back troubles, and many have reported substantial improvement. This leads me to add the thought that although successful self-treatment is gratifying, it removes some of the safeguards in the rehabilitation process. If you are an owner of a troubled back and are not a clinician, I remind you always to consult a phYSician at the outset to rule OLlt any possible existence of a condition such as a tumor or a contraindication for exercise. Acknowledgments No one will know the effort involved in writing a book until they have done so themselves. Many people have been part of the team. I specifically want to thank and acknowledge my editors at Human Kinetics Publishers: Loarn Robertson for his early suggestions and, in particular, Elaine Mustain for her expertise in the English language, ability to grasp mechanical and medical concepts, and dogged patience during the rewriting stage. Also Sylvain Grenier, my PhD student and lab manager, for the preparation of many figures that appear in this book. xv THIS PAGE INTENTIONALLY LEFT BLANK Scientific Foundation The very best professionals with expertise in low back injury prevention and rehabilitation have two things in common: an insight and wisdom developed from experience and a strong scientific foundation to enable evidence-based practice. Each problematic back is part of a whole person, who in turn is the sum of influences that range from those at the cellular level to those at the societal level. Some refer to this as the biopsychosocial approach. No two problematic backs (or patients) are the same, suggesting that optimal intervention would be different for each one. The best approach for optimal rehabilitation and prevention of those factors that exacerbate bad backs will result from wise and scientifically based decisions. Part I of this book provides a foundation for back professionals to enable them to make better decisions. THIS PAGE INTENTIONALLY LEFT BLANK Introduction to the Issues There is no sholtage of manuals and books offering wisdom on low back health. Authors range from those with formal medical or rehabilitation training to laypeople who have found an approach to alleviate their own back troubles and become self-proclaimed low back health prophets. Their intentions are honorable, but their advice is rarely based on a sound scientific foundation. Too many of these authors offer inappropriate recommendations or even harmful suggestions. Years ago, as I began to develop scientific investigations into various aspects of low back problems, I would ask my graduate students to find the scientific foundation for many of the "commonsense" recommendations I was hearing both in the clinic and in industrial settings. To my surprise they often reported that the literature yielded no, or very thin, evidence (note that I choose my students carefully and that they are very competent and reliable). Examples of such thinly supported "commonsense" recommendations include the following: • Bend the knees to perform a sit-up. • Bend the knees and keep the back straight to perform a lift. • Reduce the load being handled in order to reduce the risk of back troubles. In fact, each of these recommendations may be appropriate in some situations but, as will be shown, not in all. The famous economist John Kenneth Galbraith was well-known for demonstrating that actions based on common wisdom, at least in economic terms, were often doomed to fail. He stated that common wisdom is generally neither common nor wise. Galbraith eloquently expressed exactly what I had experienced with "clinical wisdom" pertaining to the low back. Many attempts at preventing low back troubles and rehabilitating symptomatic ones have

failed simply because they relied on ill-conceived clinical wisdom. This history of failed attempts is particularly unfortunate because it has lent credence to the assertions of a number of increasingly well-known "authorities" that low back injury prevention and rehabilitation programs are a waste of resources. These authorities claim that the majority of low back problems are not organic at allthat, for example, most of these difficulties have materialized because workers are 3 4 Low Back Disorders paid too much for injury compensation, have been subject to psychosocial influences, or crave sympathy. These dismissals of back injury are not justified. Back injury prevention and rehabilitation programs with strong scientific foundations, executions, and follow-up can be effective. Having stated this, I must acknowledge that justifying improved practice on scientific evidence is a dynamic process. With new evidence, the foundation will change. ' To account for such inevitable shifts, I have developed a balanced approach in these pages, reviewing the assets and liabilities and opposing views of an argument where appropriate. But fair warning! As you read this book, be prepared to challenge current thoughts and rethink currently accepted practices of injury prevention and approaches to rehabilitation. This chapter will familiarize you with some of the debatable issues regarding low back function, together with some opinions that rehabilitation professionals hold about patients, diagnosis, compensation, and disability. It will also explore the circumstances that lead to back injury and discuss the need to apply this knowledge to improve low back injury prevention and rehabilitation. Legislative Landscape: The Unfortunate Adverse Impact on Bad Backs Although most legislation and legal aCtIVIty involving bad backs are enacted with good intentions, much is counterproductive. A good example is the issue of spine range of motion (ROM). The American Medical Association (AMA) guidelines (1990), for quantifying the degree of back disability are based mostly on loss of spine ROM. Lawyers and compensation boards who need numbers for the purpose of defining disability and rewarding compensation have latched onto spine ROM as an objective and easily measured factor. In the legal arena, therapy is considered successful when the ROM has been restored or at least improved. Scientific evidence suggests, however, that after back injury, many people do not do well with an emphasis on enhancing spine mobility. In some cases, back problems are actually exacerbated by this approach. In fact, evidence shows that many back injuries improve with stabilizing approaches-motor control training, enhancement of muscle endurance, and training with the spine in a neutral position (Saal and Saal, 1989, may be considered the classic work). Our most recent work has shown that three-dimensional low back range of motion has no correlation to functional test scores or even the ability to perform occupational work (Parks et al., in press). In the best practice, spine flexibility may not be emphasized until the velY late stages of rehabilitation, if ever. How, then, did this idea of flexibility as the best measure of successful rehabilitation become so entrenched? The current metric for determining disability appears to have been chosen for legal convenience rather than for a positive impact on low back troubles. The current landscape creates a reward system for therapy that arguably hinders optimal rehabilitation. Perhaps the criteria for determining disability need to be reassessed and justified with scientific evidence. Another example illustrates the perverse impact of well-intended equity legislation. We all have equal rights under the law, but we are not physical equals. Although individual variance is present in every group, different populations within society demonstrate quite different capabilities. For example, the data of Jager and colleagues (1991), compiled from many studies, clearly showed that young men can tolerate more compressive load down their lumbar spines than can older men, and similarly, men can tolerate about an additional third more load than can women when matched for age. Yet human rights legislation, which is designed to create

fairness and equity Introduction to the Issues 5 by discouraging distinctions among groups, actually puts older females at greater risk than younger men. By not allowing a 64-year-old osteoporotic woman to be treated (and protected) differently from a 20-year-old, fit, 90-kg (200-lb) male in terms of tolerating spine load, the legislation presents a major barrier for intelligently implementing tolerance-based guidelines for protecting workers. Deficiencies in Current Low Back Disorder Diagnostic Practices It is currently popular for many authorities to suggest that back trouble is not a medical condition. They assert that physical loading has little to do with low back injll1Y compensation claims; rather, they believe workers complain of back problems in order to benefit from overly generous compensation packages or to convince physicians they are sick. According to this view, any biomechanically based injury prevention or rehabilitation program is useless. Variables within the psychosocial sphere dominate any biological or mechanical variable. If this is true, then this book is of no value-it should be about psychosocial intervention. Those who contend that psychosocial factors dominate low back issues are wellpublished scientists and physicians. For example, Professor Richard Deyo (1998) summarized the view of many: "Consider the following paradox. The American economy is increasingly postindustrial, with less heavy labor, more automation and more robotics, and medicine has consistently improved diagnostic imaging of the spine and developed new forms of surgical and nonsurgical therapy. But work disability caused by back pain has steadily risen." This line of reasoning assumes that modern work (i.e., more repetitive, more sedentary) is healthier for the back than the predominantly physical labor of past generations. The evidence suggests, however, that the repetitive motions required by some speCialized modern work or the sedentalY nature characteriZing others, produce damaging biomechanical stressors. In fact, the variety of work performed by our great-grandparents may have been far healthier. Deyo also seems to assume that nonsurgical therapy has been appropriately chosen for each individual, whereas I suggest that inappropriate therapy prescriptions remain quite common. Professor Alf Nachemson (1992) wrote that "most case control studies of crosssectional design that have addressed the mechanical and psychosocial factors influencing LBP (low back pain), including job satisfaction, have concluded that the latter play a more important role than the extensively studied mechanical factors." Yet none of the several references cited to support this opinion made reasonable quantification of the physical job demands. Generally, these studies found that psychosocial variables were related to low back troubles, but in the absence of measuring mechanical loading, they had no chance to evaluate a loading relationship. Finally, Dr. Nortin Hadler (2001) has been rather outspoken, stating, for example, that "it is u nclear whether there is any meaningful association between task content and disabling regional musculoskeletal disorders for a wide range of physical disorders" and that "on the other hand, nearly all multivariate cross sectional and longitudinal studies designed to probe for associations beyond the physical demand of tasks, detect associations with the psychosocial context of working." Recent evidence clearly shows that, although psychosocial factors can be important in modulating patient behavior, biomechanical components are important in leading to low back disorders and in their prevention. The position that biomechanics plays no role in back health and activity tolerance can be held only by those who have never performed physical labor and have not experienced firsthand the work methods 6 Low Back Disorders that must be employed to avoid disabling injury. While the scientific evidence is absolutely necessary, it will only confirm the obvious to those who have this experience. I find it perversely satisfying when physicians tell me that they are now, after missing work as a result of a nasty back episode related to physical work, able to relate to their patients. Perhaps experience with a variety of

heavy work and with disabling pain should be required for some medics! It is, then, essential to investigate and understand the links among loading, tissue damage or irritation, psychosocial factors, and performance to provide clues for the design and implementation of better prevention and rehabilitation strategies for low back troubles. In the following sections I will address several commonly held beliefs about back injury. Is It True That 85% of Back Troubles Are of Unknown Etiology? Low back injUly reports often mention the statistic that 85% of low back troubles are of unknown etiology. This has led to the popular belief that disabling back troubles are inevitable and just happen, a statement that defies the plethora of literature linking specific mechanical scenarios to specific tissue damage. Some have argued that this statement is simply the product of poor diagnosis-or of clinicians reaching the end of their expertise (e.g., Finch, 1 999). In fairness I must point out that diagnosis often depends on the profession of the diagnostician. Each group attempts to identify the primaly dysfunction according to its palticular type of t.reatment. For example, a physical therapist will attempt diagnosis to guide decisions regarding manual therapy approaches, while a surgeon may find a diagnOSis directed toward making surgical decisions more helpful. Some clinicians (surgeons, for example) seek a specific tissue as a pain candidate. From this perspective, nerve block procedures have shown conclusive pain source diagnosis in well over 50% of cases (e.g., Bogduk et a!., 1996; Lord et a!. , 1996; Finch, 1 999). This has prompted research into which tissues are innervated and are candidates as pain generators. Biomechanists often argue that this may be irrelevant since a spine with altered biomechanics has altered tissue stresses. Thus a damaged tissue may cause overload on another tissue causing pain whether the damaged tissue is innervated or not. This is why other clinicians employ skilled provocative mechanical loading of specific tissues to reveal those that hurt or at least to reveal loading patterns or motion patterns that cause pain. These types of functional diagnoses are helpful in designing therapy and in developing less painful motion patterns, but the process of functional diagnoses will be hindered with a poor understanding of spine biomechanics. Furthermore, those with a thorough understanding of the biomechanics of tissue damage can be guided to a general diagnosis by reconstructing the instigating mechanical scenario. An additional benefit of this approach is that once the cause is understood, it can be removed or reduced. Unfortunately, many patients continue to have troubles simply because they continue to engage in the mechanical cause. Familiarity with spine mechanics will dispel this myth of undiagnosable back trouble and reduce the percentage of those with back troubles of no known cause. Even with a tissue-based diagnosis, the practice of treating all patients with a specific diagnOSiS with a Singular therapy has not proven productive (Rose, 1989). For example, success rates with many cancer therapies greatly improved with the combination of chemotherapy and radiotherapy. Optimal back rehabilitation requires removal of the cause and the addition perhaps of stability, manual soft tissue therapy, or something else depending on the patient. Few patients fall into a "complete fit" for functional diagnosis where a Singular approach will yield optimal results. Both for interpretation of the literature and for clinical decision making, it would appear prudent to question the diagnostic criteria needed before a given diagnOSiS is assigned. Introduction to the Issues 7 Limitations in tissue-based diagnosis should not be used to suggest that determining the cause of back troubles is irrelevant nor that the manual/medical treatment in some cases is fru itless, leaving psychosocial approaches to prevail by default. Even given the current limitations, the diagnostic approach is productive for guiding prevention and rehabilitation approaches. Is It True That Most Chronic Back Complaints Are Rooted in Psychological Factors? ' While there is no doubt that many chronic back cases have psychological overlays, the significance of psychology for back

problems is often greatly exaggerated. Dr. Ellen Thompson (1997) coined the phrase "bankrupt expertise" when referring to spine docs who are unable to guide improvement in their patients and default to blaming the patients and their psychoses. These physiCians either dismiss mechanical causation or assume that mechanical causation has been adequately addressed. At our university clinic I see patients who have been referred by physicians for consult. These are either elite performers or the very difficult chronic bad backs who have failed with all other approaches. In spite of the fact that these people have received very thorough attention, I am continually heartbroken to hear about the minimal notice paid to ongoing back stressors and of the exercises that these "basket case backs" have been prescribed that have only exacerbated their condition. The day before I wrote this section, I saw a classic example. A woman had suffered for five years on disability and had seen no fewer than 12 specialists from a variety of disciplines. Although several had acknowledged that she had physical concerns, her troubles were largely attributed to mental depression. She consistently reported being unable to tolerate specific activities while being able to tolerate others. Some provocative testing confirmed her report together with uncovering a previously undiagnosed arthritic hip. For years she had been faithfully following the instructions of her health care providers to perform pelvic tilts, knees-to-chest stretches first thing in the morning, and sit-ups; to take her large dog for walks; and so on. All of these ill-chosen suggestions had prevented her posterior disc (with sciatica)-based troubles from improving. As we will see later, these types of troubles typically do not recover with flexion-based approaches-particularly first thing in the morning. Moreover, the lead-irrJ3bsed torsional loads that she experienced every time she walked her dog exceeded her tolerance. Although she reported vacuuming as a major exacerbator of her troubles, her health care providers had never shown her how to vacuum her home in a way to spare her back. I suggested that removing these daily activities and replacing the flexion stretches with neutral spine pOSition awareness training and isometric torso challenges would likely start a slow, progressive recovelY pattern. I believed that her psychological concerns would probably disappear with her back symptoms if she fell into the typical pattern. This patient, with this typical story, has a reasonably good chance to enjoy life once again. (Note: this patient was back to work and off her antidepressant medication at the time of proofing this manuscript.) None of the "experts" this woman had seen-including physical therapists, chiropractors, psychologists, physiatrists, neurologists, and orthopods-addressed mechanical concerns. This is not to condemn these professions but rather to suggest that sharing experiences and approaches will help us to be more successful in helping bad backs. Perhaps these professionals were unaware of the principles of spine function, the types of loads that are imposed on the spine tissues during certain activities, and how these activities and spine postures can be changed to greatly reduce the loads-in other words, the biomechanical components. 8 Low Back Disorders This book is an attempt to heighten the awareness and potential of this mechanical approach. While it sounds velY harsh, I have found relatively few experts who appear willing to adequately address the causes of back troubles while working to find the most appropriate therapy. My years of laboratoly-based work combined with collaboration in recent years with my clinical colleagues have provided me with unique insight. As a result, I am not so quick to blame the chronic patient. Does Pain Cause Activity Intolerance? Evidence that mechanical tissue overload causes damage is conclusive. But does the damage cause pain, and does the chronic pain cause work intolerance? Several, but limited numbers of, studies have mechanically or chemically stimulated tissues to reproduce clinical pain patterns. (The absence of definitive, large-scale studies is due to the ethical issues of performing invasive procedures and probably not because such studies are

without scientific merit.) For example, the pioneering work of Hirsch and colleagues (1963-64) documented pain from the injection of hypertonic saline into specific spine tissues thought to be candidates for damage. Subsequent to this work, several other studies suggested the link between mechanical stimulation and pain, for example, the work of Hsu and colleagues (1988), documenting pain in damaged discs. There is irrefutable evidence that vertebral disc end-plate fractures are very common and only result from mechanical overload (Brinckmann, Bigemann, and Hilweg, 1 989; Gunning, Callaghan, McGill, 2001). That these fractures are also found in necropsy specimens that were subjected to whiplash (Taylor, Twomey, and Corker, 1 990) also strengthens another facet of this relationship. Hsu performed discograms (injections of radio contrast) into 692 discs of which 14 demonstrated leakage into the vertebral body, confirming an end-plate fracture. Four of these discs (28%) produced severe pain, nine (64%) produced fully concordant pain, and one produced mildly discordant pain. In contrast, only 1 1% of the remaining 678 discs with no end-plate disruption produced severe pain, 31% with corkgrdant pain, 1 7% with mild pain, and 41% with no pain. This evidence provides strong support for the notion that loading causes damage and damage causes pain. Even though pain can limit function and activity in other areas of the body, some still suggest that these are not linked when a bad back is at issue. Teasell (1997) provided an interesting perspective when he argued that in sports medicine, as opposed to occupational medicine, it is well accepted that some injuries require months of therapy or can even cause retirement from the activity. He noted that athletes receiving specialized sports medicine care are an interesting group to consider since many are highly motivated, are in top physical condition, are well paid, have access to good medical care, and are fully compensated even while injured. Their injuries and pain can cause absence from play for substantial amounts of time and can even end their lucrative careers. Teasell reminded us that not all long-term chronic pain is an entirely psychosocial concern, as implied by some clinicians. These clinicians' dismissal of the usefulness of physical approaches simply because they have not been successful in reducing long-term troubles is a disservice to the patient. Inadequacies in Current Care and Prevention of Low Back Disorders Many back patients can testify that the care they have received for their troubles is not satisfactory. What are some of the factors that contribute to the inadequacy of their experience? Certainly one of them is the fact that the epidemiological evidence on Introduction to the Issues 9 which many professionals base their treatment recommendations can be quite confusing. Following are some examples of the issues that cause confusion. • Plethora of studies on "backache." NonspeCific "backache" is nearly impossible to quantify and, even if it could be quantified, offers no guidance for intervention. As such, any study of treatment interventions on nonspecific backache is of little use. Some backs suffer with discogenic problems, for example, and will respond quite differently from those with ligamentous damage or facet-based problems. Efficacy studies that do not subclassify bad backs end up with nonspeCific "average" responses. This has led to the belief that nothing works-or that evelything does, but to a limited degree. More studies on nonspecific backache treatment will not be helpful, nor will the large epidemiological reviews of these studies offer real insight. /' • V-shaped function of loading and resulting injury risk. Like many healthrelated phenomena, the relationship of low back tissue loading to injury risk appears to form a U-shaped function-not a monotonically rising line. For example, virtually every nutrient will cause poisoning with excessive dosage levels, but health suffers in their absence; thus, there is a moderate optimum. In the case with low back loading, evidence suggests that two regions in the U-shaped relationship are problematic-too much and too little. Porter (987) suggested that heavy work is good for the backbut how does one define

heavy? Porter was probably referring to work of sufficient challenge and variability to reach the bottom of the U and hence lower symptoms. From a biological perspective, sufficient loading is necessalY to cause strengthening and toughening of tissues, but excessive levels will result in weakening. This lack of consensus in the literature regarding the measurement of exposure has been problematic (Marras et aI. , 2000). A more advanced understanding is required. • Relationship of intensity, duration of loading, and rest periods. As Ferguson and Marras (997) pointed out, some studies suggest that a certain type of loading is not related to pain, inj LI1Y , or disability, while others suggest it is, depending on how the exposure was measured and where the moderate optimum for tissue health resides for the experimental population. The subjectivity of such studies is further underscored when we consider the question, Is there a clinical difference between tissue irritation and tissue damage? Loading experiments on human and animal tissues to produce damage reveal the "ultimate tolerable load" beyond which injuries cause biomechanical changes, pain, and gross failure to structures. In real life, any of us could irritate tissues to produce tremendous pain at loading levels well below the cadavericly determined tolerance by repeated and prolonged loading. In fact, evidence presented by Videman and colleagues (995) suggests that the progressive development of conditions such as spinal stenosis results from years of specific subfailure activity. The fundamental question is, Could such conditions be avoided by evidence-based prevention strategies that include optimal loading, rest periods, and controlling the duration of exposure? III-Advised Rehabilitation Recommendations These failures to frame research and its results appropriately have resulted in many oversimplifications about low back treatment, which have in turn led to some inadequate treatment practices and recommendations. A few of the most common recommendations for back health are discLlssed here. • Strengthen muscles in the torso to protect the back. Despite the clinical emphasis on increasing back muscle strength, several studies have shown that muscle strength cannot predict who will have future back troubles (Biering-Sorenson, 1984). On the other hand, Luoto and colleagues (995) have shown that muscular endurance 10 Low Back Disorders (as opposed to strength) is protective. Why, then, do many therapeutic programs continue to emphasize strength and neglect endurance? Perhaps it is a holdover influence from the athletic world in which the goal of training is to enhance performance. Perhaps it is an influence from the pervasive use of bodybuilding approaches in rehabilitation. As will be shown, optimal exercise therapy occurs when the emphasis shifts away from the enhancement of performance and toward the establishment of improved health. In many cases the two are mutually exclusive! • Bend the knees when performing sit-ups. Clinicians widely recommend bending the knees during a sit-up, but on what evidence? A frustrating l iterature search suggests that this perception may be the result of "clinical wisdom." Interestingly, Axler and McGill (997) demonstrated that there is little advantage to one knee position over the other, and in fact the issue is probably moot because there are far better ways to challenge the abdominal musculature and impose lower lumbar spine loads (traditional sit-ups cause spine loading conditions that greatly elevate the risk of injury). This issue is one of many that will be challenged in this book. • Performing sit-ups will increase back health. Is this a true statement or an artifact of experimental methodology? Despite what many would like to believe, there is only mild literature support for the belief that people who are fit have less back trouble (although positive evidence is increasing, for example, Stevenson et al., 2001). Interestingly, many of the studies attempting to evaluate the role of increased fitness in back health actually included exercises that have been known to cause back troubles in many people. For example, many have attempted to enhance abdominal strength with sit-ups. After examining the lumbar compression that results from performing

situps with full flexion of the lumbar spine, it is clear that enough sit-ups will cause damage in most people. Each sit-up produces low back compression levels close to the National Institute for Occupational Safety and Health (NIOSH) action limit, and repeatedly compressing the spine to levels higher than the NIOSH action limit has been shown to increase the risk of back disorders (Axler and McGill, 1997). Thus, reaching a conclusion over the role of fitness from the published literature has been obscured by ill-chosen exercises. Increased fitness does have support, but the way in which fitness is increased appears to be critical. • To avoid back injury when lifting, bend the knees, not the back. Probably the most common advice given by the clinician to the patient who must lift is to bend the knees and keep the back straight. In addition, this forms the foundation for virtually evelY set of ergonomic guidelines provided to reduce the risk of work-related injury. Very few jobs can be performed this way. Further, despite the research that has compared stooping and squatting styles of lifting, no conclusion as to which is better has been reached. The issue of whether to stoop or squat during,a lift depends on the dimensions and properties of the load, the characteristics of the lifter, the number of times the lift is to be repeated, and so forth, and there may in fact be safer techniques altogether. Much more justifiable guidelines will be developed later in the text. • A single exercise or back stability program is adequate for all cases. It is currently popular to promote the training of single muscles to enhance spine stability. While the original research was motivated by the intention to reeducate perturbed motor patterns that were documented to be the result of injury, others have misinterpreted the data and are promoting exercises to train muscles they believe are the most important stabilizers of the spine. Unfortunately, they did not quantify stability. The process of quantifying the contribution of the anatomical components to stability reveals that virtually all muscles can be important, but their importance continually changes with the demands of the activity and task. It is true that damage to any of the spinal tissues from mechanical overload results in unstable joint behavior. Because of I ntroduction to the Issues 11 biomechanical changes to the joint, however, the perturbed tissue is rarely linked to the symptomatology in a simple way. More likely other tissues become involved, and which ones are involved will result in different accompanying motor disturbances. This variety in possible etiologies means that a single, simple rehabilitation approach often will not work. Is mobility to be restored at the expense of normal joint stability? Or is stability fO be established first with enhanced mobility as a secondary delayed rehabilitation goal? Or is the clinical picture complex, for example, in a situation in which spine stability is needed but tonic psoas activity causing chronic hip flexure necessitates hip mobilization? This example, one of many that could have been chosen, illustrates the challenge of ensuring sufficient stability for the spinal tissues. No simple, or single, approach will produce the best results in all cases. The description and data presented in this book will help guide the formulation of exercises that ensure spine stability. Can Back Rehabilitation Be Completed in 6 to 12 Weeks? Some have suggested that damaged tissues should heal within 6 to 12 weeks. In fact, many have used this argument to suppOtt the notion that work intolerance exceeding this period has no pathoanatomical basis (e.g., Fordyce, 1 995) but stems from psychosocial issues. Further, some have suggested that patient recovery would be better served by redirecting rehabilitative efforts away from physical approaches. This position can be refuted by data and indicates a misunderstanding of the complexities of spine pathomechanics. The concept that tissues heal within 6 to 12 weeks appears to be originally based on animal studies (reviewed in Spitzer, 1 993). However, not all human patients get better so quickly (Mendelson, 1982), and the follow-up studies from some defined disorders such as whiplash are compelling in the support of lingering tissue disruption (e.g. , Radanov et al., 1 994).

Evidence will be presented in later chapters of both mechanical and neurological changes that linger for years subsequent to injury. This includes loss of various motor control parameters together with documented asymmetric muscle atrophy and other disorders. This suggests that postinjury changes are not a simple matter of gross damage "healing." Following are only a few of the types of damage that can be long term indeed: • Specific tissues such as ligaments, for example, have been shown to take years to recover from relatively minor insult (Woo, Gomez, and Akeson, 1 985), • The intervertebral motion units form a complex mechanism involving intricate interplay among the parts such that damage to one part changes the biomechanics and loading on another part. From the perspective of pathomechanics, many reports have documented the cascade of biomechanical change associated with initial disc damage and subsequent joint instability and secondary arthritis, which may take years to progress (e.g., Brinckmann, 1 985; Kirkaldy-Willis, 1 998). • Videman and colleagues (995) documented that vertebral osteophytes were most highly associated with end-plate irregularities and disc bulging. Osteophytes are generally accepted to be secondary to disc and end-plate trauma but take years to develop. Thus, to suggest that back troubles are not mechanically based if they linger longer than a few months only demonstrates a limited expertise. Another question is, Can these back troubles linger for a lifetime? In this connection, it is interesting that elderly people appear to complain about bad backs less than younger people. Valkenburg and Haanen (982) showed that back troubles are more 12 Low Back Disorders frequent during the younger years. Weber (1983) provided fUlther insight by reporting on patients who were 10 years post disc herniations (some of them had had surgery while others had not) and were engaged in strenuous daily activity-yet all were still receiving total disability benefits! It would appear that the cascade of changes resulting from some forms of tissue damage can take years but generally not longer than 10 years. Although the bad news is that the affected joints stiffen during the cascade of change, the good news is that eventually the pain is gone. To summarize, the expectation that damaged low back tissues should heal within a matter of weeks has no foundation. In fact, longer-term troubles do have a substantial biomechanical/pathoanatomical basis. On the other hand, troublesome backs are generally not a life sentence. Should the Primary Goal of Rehabilitation Be Restoring the Range of Motion? Research has shown that an increased range of motion in the spine can increase the risk of future back troubles (e.g. , Battie et al., 1990; Biering-Sorenson, 1 984; Burton, Tillotson, and Troup, 1 989). Why then does increasing the range of motion remain a rehabilitation objective? The first reason, discussed earlier, is the need to quantify reduced disability as defined by the AMA. Second, there is a holdover philosophy from the athletic world that increased range of motion enhances performance. This may be true for some activities, but it is untrue for others. As will be shown, this philosophy may work for other joints, but it generally does not work for the back. In fact, successful rehabilitation for the back is generally retarded by following athletic principles. Mechanical loading and the Process of Injury: A Low Back Tissue Injury Primer Any clinician completing a worker/patient compensation form is required to identify the event that caused the injury. Very few back injuries, however, result from a single event. This section documents the more common cumulative trauma pathway leading to the culminating event of a back injLI1y. Because the culminating event is falsely presumed to be the cause, prevention efforts are then focused on that event. This misdirection of efforts fails to deal with the real cause of the cumulative trauma. While a generic scenario for injury is presented here, chapter 4 offers a more indepth discussion of injLI1Y from repeated and prolonged loading to specific tissue. The purpose of this section is to promote consideration of the many factors that modulate the risk of tissue failure and to encourage probing to generate

appropriate hypotheses about injury etiology. Injury, or failure of a tissue, occurs when the applied load exceeds the failure tolerance (or strength of the tissue). For the purposes of this discussion, injury will be defined as the full continuum from the most minor of tissue irritation (but microtrauma nonetheless) to the grossest of tissue failure, for example, vertebral fracture or ligament avulsion. We will proceed on the premise that such damage generates pain. Obviously, a load that exceeds the failure tolerance of the tissue, applied once, produces injury (see figure 1 . 1b in which a Canadian snowmobiler airborne and about to experience an axial impact with the spine fully flexed is at risk of posterior disc herniation upon landing). This injury process is depicted in figure 1.1a, where a margin of safety is observed in the first cycle of subfailure load. In the second loading cycle, the applied load increases in magnitude, simultaneously decreasing the margin of safety to zero, at which point an injury occurs. While this description of low back injury is common, particularly among medical practitioners who are required to identify Load a Failure tolerance i Margin of safety Time b Introduction to the Issues 1 3 Figure 1 .1 (a) A margin of safety is observed in the first cycle of subfailure load. In the second loading cycle, the applied load increases in magnitude, simultaneously decreasing the margin of safety to zero, at which point an injury occurs. (b) The Canadian snowmobile driver (the author in this case who should know better) is about to experience an axial compressive impact load to a fully flexed spine. A one-time application of load can reduce the margin of safety to zero as the applied load exceeds the strength or failure tolerance of the supporting tissues. Reprinted from Journal of Biomechanics, 30 (5), S.M. McGill,"Invited paper: Biomechanics of low back injury: Impl ications on current practice and the clinic," 456-475, 1 997, with permission from Elsevier Science. Load a an injUly-causing event when completing forms for workers' compensation reports, my experience suggests that relatively few low back injuries occur in this manner. More commonly, injury during occupational and athletic endeavors involves cumulative trauma from repetitive subfailure magnitude loads. In such cases, injury is the result of accumulated trauma produced by either the repeated application of relatively low load or the application of a sustained load for a long duration (as in a sitting task, for example). An individual loading boxes on a pallet who is repeatedly loading the tissues of the low back (several tissues could be at risk) to a subfailure level (see figure 1 .2, a-b) Failure tolerance Failure Margin of safety Time Figure 1 .2 (a) Repeated subfailure loads lead to tissue fatigue, reducing the failure tolerance, leading to failure on the Nth repetition of load, or box lift in this example (b). Reprinted from Journal of Biomechanics, 30 (5), S.M. McGill,"Invited paper: Biomechanics of low back injury: Implications on current practice and the clinic," 456-475, 1 997, with permission from Elsevier Science. 14 Low Back Disorders a experiences a slow degradation of failure tolerance (e.g., veltebrae, Adams and Hutton, 1 985; Brinckmann, Biggemann, and Hilweg, 1 989). As tissues fatigue with each cycle of load and correspondingly the failure tolerance lowers, the margin of safety eventually approaches zero, at which point this individual will experience low back injury. Obviously, the accumulation of trauma is more rapid with higher loads. Carter and Hayes (1977) noted that, at least with bone, fatigue failure occurs with fewer repetitions when the applied load is closer to the yield strength. Yet another way to produce injury with a subfailure load is to sustain stresses constantly over a period of time. The rodmen shown in figure 1 .3b, with their spines fully flexed for a prolonged period of time, are loading the posterior passive tissues and are initiating time-dependent changes in disc mechanics (figure 1 .3a). Under sustained loads these viscoelastic tissues slowly deform and creep. The sustained load and resultant creep causes a progressive reduction in the tissue strength. Correspondingly, the margin of safety also declines until injury occurs at a specific percentage of tissue strain (i.e.,

at the breaking strain of that particular tissue). Note that these workers are not lifting a heavy load; simply staying in this posture long enough will eventually ensure injurious damage. The injury may involve a single tissue, or a complex picture may emerge in which several tissues become involved. For example, the prolonged stooped posture imposes loads on the posterior ligaments of the spine and posterior fibers of the intervertebral disc. The associated creep deformation that ultimately produces microfailure (e.g., Adams, Hutton, and Stott, 1 980; McGill and Brown, 1 992) may initiate another chain of events. Stretched ligaments increase joint laxity, which can lead to hyperflexion injury (to the disc) and to the following sequence of events: 1. Local instability 2. Injl\1y of unisegmental structures 3. Ever-increasing shearing and bending loads on the neural arch This laxity remains for a substantial period after the prolonged stoop. Failure tolerance Failure Load j Margin of safety l Applied load b Time Figure 1 .3 (a) These rodmen with fully flexed lumbar spines are loading posterior passive tissues for a long duration, (b) reducing the failure tolerance leading to failure at the Nth% of tissue strain. Tolerance Applied load Rest Time Figure 1 .4 Loading is necessary for optimal tissue health. When loading and the subsequent degradation of tolerance are followed by a period of rest, an adaptive tissue response increases tolerance. Tissue "training" results from the optimal blend of art and science in medicine and tissue biomechanics. Introduction to the Issues 1 5 Understanding the process of tissue damage in th\s way em\:lhasizes why sin"l\:lle i.n.\ury \:lrevention approaches often fail. More effective injury intervention strategies recognize and address the complexities of tissue overload. Avoidance of loading altogether is undesirable. The objective of injury prevention strategies is to ensure that tissue adaptation stimulated from exposure to load keeps pace with, and ideally exceeds, the accumulated tissue damage. Thus, exposure to load is necessary, but in the process of accumulating microtrauma, the applied loads must be removed (with rest) to allow the healing/adaptation process to gradually increase the failure tolerance to a higher level. We have already seen how tissue loading and injury risk form an optimal U-shaped relationship of not too much and not too little load. Detennining the optimal load for health encompasses both the art and science of medicine and tissue biomechanics. Figure 1 .4 presents a final load-time history to demonstrate the links among loading, rest, and adaptive tissue tolerance. In summary, the injury process may be associated either with very high loads or with relatively low loads that are repeated or sustained. This either/or causation necessitates rigorous examination of injury and tissue loading history for substantial periods of time prior to the culminating injury event. It is important to recognize that simply focusing on a single variable such as one-time load magnitude may not result in a successful index of risk of injury, particularly across a w ide variety of activities. A Final Note The selected controversies introduced in this chapter illustrate the need for the evide1Ke presented in the rest of the text and the relevance of the discussions that follow. Resist the urge to assume that conventional wisdom is correct: first consider the evidence and then form your own opinions. References Adams, M.A., and Hutton, w.e. (985) Gradual disc prolapse. Spine, 10: 524. Adams, M .A., Hutton, W.e., and Stott, JR.R. (980) The resistance to flexion of the lumbar intervertebral joint. Spine 5: 245. American Medical Association. (1990) Guides to the evaluation of penm1l1ent impairment C 3rd edition} Axler, c.T. , and McGill, S.M. (1997) Choosing the best abdominal exercises based on knowledge of tissue loads. Medicine and Science in Sports and Exercise, 29: 804-81l. Battie, M.C., Bigos, S.]., Fisher, L.D., Spengler, D.M., Hansson, T.H., Nachemson, A.L., and Wortley, MD. (1 990) The role of spinal flexibility in back pain complaints within industry: A prospective study. Spine, 15: 768-773. Biering-Sorensen, F. (1984) Physical measurements as risk indicators for low-back trouble over a one-year period. Spine, 9: 1

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Weber, CL., Smith,].T., Dumas, G.A., and Albert, w.]. (2001) A longitudinal study of the development of low back pain in an industrial population. Spil1e, 26 (1 2): 1 370- 1 377. Taylor,].R., Twomey, L.T., and Corker, M. (990) Bone and soft tissue injuries in post-mortem lumbar spines. Paraplegia, 28: 1 19-129. Introduction to the Issues 17 Teasell, R.W. (1997) The denial of chronic pain. Journal of Pain Research Management, 2: 89-91. Thompson, E.N. (199Y:l Back pain: Bankrupt expertise and new directions. Journal of Pain Research Management, 2: 1 95-196. Valkenburg, H.A., and Haanen, H.e.M. (1982) The epidemiology of low back pain. In: White, A.A., and Gordon, S.L. (Eds.), Symposium on idiopathic low back pain. St. Louis: Mosby. Videman, T. , Battie, M.e., Gill, K., Manninen, H., Gibbons, L.E., and Fisher, L.D. (1995). Magnetic resonance imaging findings and their relationships in the thoracic and lumbar spine: Insights into the etiopathogenesis of spinal degeneration. Spine, 20 (8): 928-935. Weber, H. (1983) Lumbar disk herniation: A controlled prospective study with ten years of observation. Spine, 8: 131. Woo, S.L.-Y. , Gomez, M.A., and Akeson, W.H. (1985) Mechanical behaviors of soft tissues: Measurements, modifications, injuries, and treatment. In: Nahum, H.M., and Melvin, J. (Eds.), Biomechanics ()/trauma (pp. 109-133). Norwalk, CT: Appleton Century Crofts. THIS PAGE INTENTIONALLY LEFT BLANK Scientific Approach Unique to This Book This book contains many nontraditional viewpoints on how the spine functions and becomes damaged. Most of these perspectives have emerged from a unique biomechanically based methodological approach. This chapter will familiarize you with the unique general approach taken for obtaining much of the data in this book and will help you begin to understand both the limitations and the unique insights prOVided by such an approach. As spine biomechanists, our methods of inquiry are similar to those used by mechanical or civil engineers. For example, a civil engineer charged with the task of building a bridge needs three types of information: • Traffic to be accommodated, or the design load • Structure to be used (e.g., space truss or roman arch), as each architecture possesses specific mechanical traits and features • Characteristics of proposed materials that will affect strength, endurance, stability, resistance to structural fatigue, and so on. Our approach to investigating spine function is similar to that of our engineering colleagues. We begin with the follOWing relationship to predict the risk of tissue damage: Applied load > tissue strength = tissue failure (injury) Recall from the tissue injury primer at the end of chapter 1 that tissue strength is reduced by repeated and prolonged loading but is increased with subsequent rest and adaptation. Analyzing tissue failure in this way requires two distinct methodological approaches. This is why we developed two quite distinct laboratories, which led to much of the progress documented in this book. (Please note that the "we" used in this chapter includes my research team of graduate students, visiting scholars, and technicians.) Our first lab is equipped for in-vitro testing of spines, in which we purposefully tty to create herniated discs, damaged end plates, and other tissue-specific injuries. The second lab is the in-vivo lab where living people (both normals and patients) are tested for their response to stress and loading. Individual tissue loads are obtained from sophisticated modeling procedures. 19 20 Low Back Disorders In-Vitro Lab The in-vitro lab is equipped with loading machines, an acceleration rack, tissue sectioning equipment, and an X-ray suite to document progressive tissue damage. For example, by performing discograms with radio opaque contrast liquids, we can document the mechanics of progressive disc herniation. We investigate any other injury mechanisms in the same way-that is, by a pplying phYSiological loads and motion patterns and then documenting the damage with appropriate technology. Since many technique issues can affect the experimental results, the decision to use one over another is governed by the specific research question. For example, since a matched set of human spines to run a controlled failure test cannot be obtained, animal models must be used.

Here, control is exercised over genetic homogeneity, diet, physical activity, and so fOIth to contrast an experimental cohort with a matched set of control spines. Of course, the results must be validated and interpreted for them to be relevant to humans. In addition, identifying the limitations for relevance in interpretation is critical. Some hypotheses that demand the use of human material are compromised by the lack of available young, healthy, undegenerated specimens. Having healthy specimens is critical because biomechanics and injury mechanisms radically change with age. Other major methodological issues include the way in which biological tissues are loaded, perhaps at specific load rates or at specific rates of displacement. The researcher must decide which has the most relevance to the issue at hand. Devising these experiments is not a trivial task. In-Vivo Lab The in-vivo lab is unique in its approach in attempting to document the loads on the many lumbar tissues in vivo. This knowledge lends powerful insight into spine mechanics, both of normal functioning and of failure mechanics. Since transducers cannot be routinely implanted in the tissues to measure force, noninvasive methods are necessary. The intention of the basic approach is to create a virtual spine. This virtual model must accurately represent the anatomy that responds dynamically to the threedimensional motion patterns of each test subject/patient and must mimic the muscle activation patterns chosen by the individual. In so doing, it enables us to evaluate subjects' unique motor patterns and the consequences of their choices and skill. How the Virtual Spine Works While two groups (the Marras group [e.g., Granata and Marras, 1 9931 and the McGill group) have devoted much effort to the development of biologically driven models, the McGill model will be described here given its familiarity to the author. The modela dynamic, three-dimensional, anatomically complex, biologically driven approach to predicting individual lumbar tissue loads-is composed of two distinct parts: a linkedsegment model and a highly detailed spine model that determines tissue loads and spine stability. • The first part of the McGill model is a three-dimensional linked-segment representation of the body using a dynamic load in the hands as input. Two or more video cameras at 30 Hz record joint displacements to reconstruct the joints and body segments in three dimensions. Working through the arm and trunk linkage using linkedsegment mechanics, reaction forces and moments are computed about a joint in the b a Scientific Approach Unique to This Book 21 ��====----��177- Figure 2.1 The tissue load prediction approach requires two models. The first (a) is a dynamic three-dimensional linked-segment model to obtain the three reaction moments about the low back. The second model (b) partitions the moments into tissue forces (muscle forces 1-18; ligaments 19-26; and moment contributions from deformed disc, gut, and skin in bending). low back (usually L4-L5) (previously described in McGill and Norman, 1 985) (see figure 2.1a). Using pelvic and spine markers, the three reaction moments are converted into moments about the three orthopedic axes of the low back (flexion/extension, lateral bend, and axial twist). • The second part of the McGill model enables the partitioning of the reaction moments obtained from the linked-segment model into the substantial restorative moment components (supporting tissues) using an anatomically detailed three-dimensional representation of the skeleton, muscles, ligaments, nonlinear elastic intervertebral discs, and so on (see figure 2. 1b). This part of the model was first described in McGill and Norman (1986), with full three-dimensional methods described in McGill (1992), and the most recent update provided by Cholewicki and McGill (1996). In total, 90 low back and torso muscles are represented. Very briefly, first the passive tissue forces are predicted by assuming stress-strain or load deformation relationships for the individual passive tissues. Deformations are modeled from the three-dimensional lumbar kinematics measured from the subject, which drive the vertebral kinematics of the model. Passive tissue stresses are calibrated for the differences

in flexibility of each subject by normalizing the stress-strain curves to the passive range of motion of the subject. Electromagnetic instrumentation, which monitors the relative lumbar angles in three dimensions, detects the isolated lumbar motion. The remaining moment is then partitioned among the many fascicles of muscle based on their activation profiles (measured from EMG) and their physiological cross-sectional area. The moment is then modulated with known relationships for instantaneous muscle length of either shortening or lengthening velocity. Sutarno and McGill (1995) described the most recent improvements of the forcevelOCity relationship. In this way, the modeled spine moves according to the movements of the subject's spine, and the virtual muscles are activated according to the activation measured directly from the subject (see figures 2.2, a-b and 2.3). 22 Low Back Disorders b Figure 2.2 (a) Subject monitored with EMG electrodes and electromagnetic instrumentation to directly measure three-dimensional lumbar kinematics and muscle activity. (b) The modeled spine (partially reconstructed for illustration purposes, although for the purposes of analysis it remains in mathematical form) moves in accordance with the subject's spine.The virtual muscles are activated by the EMG signals recorded from the subject's muscles. Using biological signals in this fashion to solve the indeterminacy of multiple loadbearing tissues facilitates the assessment of the many ways that we choose to suppon loads. Such an assessment is necessalY for evaluating injury mechanisms and formulating injUly-avoidance initiatives. From a clinical perspective, this ability to mimic individual spine motions and muscle activation patterns enables us to evaluate the consequences of a chosen motor control strategy. For example, we can see that some people are able to stabilize their backs and spare their lumbar tissues from overload when performing specific tasks. Conversely, we are able to evaluate the consequences of poorly chosen motor strategies. In this way we can identify those individuals with penurbed motor patterns and devise specific therapies to regroove healthy motor patterns that ensure sufficient spine stability and spare their tissues from damaging load. Our challenge has been to ensure sufficient biological fidelity so that estimations of tissue forces are valid and robust over a wide variety of activities. The three-dimensional anatomy is represented in computer memory (muscle areas are provided in appendix A.n. On occasion, if the expense is warranted, we create a virtual spine of an individual from a three-dimensional reconstruction of serial MRI slices from the hip trochanter to T4 (see figure 2. 2b). This component of the modeling process is well documented for the interested reader in McGill and Norman (1986) and McGill (1992). A list of the large number of associated research papers pertaining to the many detailed aspects of the process is provided in a separate reference section at the end of this chapter. See figures 2.4 and 2.5 for a flowchart and example of the modeling process. Scientific Approach Unique to This Book 23 Figure 2.3 In this historical photo an instrumented subject simulating the complex three-dimensional task of tossing an object.The instrumentation includes three-dimensional video to capture body segment kinematics, a recording of the three-dimensional force vector applied to the hand, a 3-SPACE electromagnetic device to record isolated threedimensional lumbar motion and assist in partitioning the passive tissue forces, and 16 channels of electromyographic electrodes to capture muscle activation patterns. Development of the Virtual Spine The development of the virtual spine approach has been an evolutionary process spanning 20 years. In that time we have had to confront several specific issues, namely, issues of validation and how to handle deep muscles that are inaccessible with surface EMG electrodes. Briefly, although we have tried intramuscular electrodes in highly selected conditions, this is a limiting invasive procedure. Generally, we estimate the deep muscle activation amplitudes from movement synergists (McGill, Juker, and Kropf, 1996). This method is limited, however,

because it requires a prior knowledge of muscle patterns for a given moment combination in a specific task. We try to incorporate the highest level of content validity by using detailed representations of the anatomy and physiological cross-sectional areas, recording stress-strain relationships, and incorporating known modulators of muscle force such as length and velocity. One of our validation exercises is to compare the three measured reaction moments with the sum of individual tissue moments predicted by the virtual spine. A close match suggests that we have succeeded in accurately representing force. Over the past five years this approach for predicting individual lumbar tissue loads has evolved to enable us to document spine stability. In this way, we can evaluate an individual's motor patterns and identify strategies that ensure safety and those that could result in injury. This is not a trivial task. It requires converting tissue forces to stiffness and using convergence algorithms to separate the forces needed to create the torques that sustain postures and movements from the additional forces needed to EMG Video ----------.I 1 Spinal model: partitioning reaction moment into individual tissues Body joint Kinematic portion: ! coordinates Orientation of rib cage and pelvis determined (3-SPACE1 Hand force 1 t Estimate lumbar vertebral position I + Linked-segment model rl Estimated center of rotation disc Three-dimensional I Memory dynamic -± Stored spacial ma trices: t Initial estimate of crude compression Pelvis and shear at L4/L5 Rib cage Reaction moment /' Each vertebra about L4/L5 Disc 1 Orient vertebrae according to estimate J 1 .-- Calculate muscle and ligament lengths and velocity of length change (dUdt) Kinetic portion: Calculate disc moment J 1 t.,. Calculate ligament stress and contribution to restorative moment I Muscle EMG t EMG AID converted F.W.R. L.E. EMG to force conversion Memory Muscle length and velocity parameters t Determine moment allocated to muscle Common gai n sted I Muscle model r I Computed muscle moments J or EMG assi optimization t • Predicted muscle moments equal to sum of muscle moments? No Yes Compute total disc compression-shear using disc model Compare all stresses (muscular, ligament, skeletal) to tolerance data Load time histories and flag tissues close to failure Figure 2.4 The model input and output are illustrated in this flowchart up to the point of calculating moments and tissue loads. Spine stability is calculated with an additional module. 24 c 0 E E '0 C/) Q) OJ Q) 0 a c 30 20 10 0 -10 -20 -30 -40 / Flexion/extension -50 -60+---,---,---,---,---,---,---,--- o 20 40 60 80 Time (s) 100 120 140 160 80 ----------. 70 60 50 30 20 Internal oblique , /I n Longissimus , ', • , , , ' I , , 10 1 ... 1 j,J\ I'\V / .,. '" \ I" ... . / Rectus '- - - v Psoas External oblique \ .... \ ... . o '·· ··· · ····· ·· · ····· · · . . .. o 20 40 60 80 100 120 140 160 Time (s) b E :co, c Q) 0.40 -r----------, Longissimus - _I 0.35 0.30 0.25 -.J 0.20 0.15 0.10 Rectus Internal oblique ..... 0.05 +--.,.---r--"T"""--.---r---...-----r---l o 20 40 60 80 1 00 120 140 160 Figure 2.S Various stages of model output in this example of a subject flexing, picking up a weight, and extending: lumbar motion about the three axes-flexion-extension, lateral bend, and axial twist (a); lengths of a few selected muscles throughout the motion (b); some muscle forces (e); L4-LS joint forces of compression and shear (d); and stability index, where larger positive numbers indicate higher stability and a zero or negative number would suggest that unstable behavior is possible (e). (continued) 25 d 26 Low Back Disorders Z Ql 2000,-------------------, 1500 ___ Axial compression 1000 10'2 1010 108 106 x Ql "0 . 104 :0 co Ci5 500 102 Medio-lateral shear 10° o --- - - - - - ---- - ._ --- -. 10-2 nterior-posterior shear 10-4 I---.,..----r----r---....,...---r--__r-----r-- -500--_r_----r__,--__r---r--....,... o 20 40 60 80 100 120 140 160 Time (s) o 20 40 60 80 100 120 140 160 Time (s) e Figure 2.S (continued). ensure stability. Potential energy-based analyses are then employed to identify the "stability index" in each degree of freedom of the joint, which in turn reveal the joint's ability to survive

a given loading scenario. This level of modeling represents the highest level of sophistication currently available. It has enabled us to challenge concepts pertaining to spine stability--more on this in chapters 6 and 13. References Cholewicki,]., and McGill, S.M. (996) Mechanical stability of the in vivo lumbar spine: Implications for injury and chronic low back pain. Clinical Biomechanics, 11 (1): 1-15. Granata, K.P., and Marras, W.S. (993) An EMG-assisted model of loads on the lumbar spine during assymetric trunk extensions. journal 0/ Biomechanics, 26: 1 429-1438. McGill, S.M. (992) A myoelectrically based dynamic 3-D model to predict loads on lumbar spine tissues during lateral bending. jou rnal o/Biomechanics, 25 (4): 395-414. McGill, S.M., and Norman, R.W. (986) The Volvo Award for 1986: Partitioning of the L4/L5 dynamic moment into disc, ligamentous and muscular components during lifting. Spine, 11 (7): 666-678. References and Bibliography Pertaining to the Finer Details of the Modeling Process Cholewicki,]., and McGill, S.M. (994) EMG Assisted Optimization: A hybrid approach for estimating muscle forces in an indeterminate biomechanical model. journal 0/ Biomechanics, 27 (0): 1287- 1289. Scientific Approach Unique to This Book 27 Cholewicki,]., and McGill, S.M. (1995) Relationship between muscle force and stiffness in the whole mammalian muscle: A simulation study. journal of Biomechanical Engineering, 1 17: 339-342. Cholewicki,]., McGill, S.M., and Norman, RW. (1995) Comparison of muscle forces and joint load from an optimization and EMG assisted lumbar spine model: Towards development of a hybrid approach. journal of Biomechanics, 28 (3): 321-331. McGill, S.M. (988) Estimation of force and extensor moment contributions of the disc and ligaments at L4/L5. Spine, 12: 1 395-1402 McGill, S.M. (996) A revised anatomical model of the abdominal musculature for torso flexion efforts. journal of Biomechanics, 29 (7): 973-977. McGill, S.M., Juker, D., and Axler, C (996) Correcting trunk muscle geometly obtained from MRI and CT scans of supine postures for use in standing postures. jou rnal of Biomechanics, 29 (5): 643-646. McGill, S.M., Juker, D., and Kropf, P. (996) Appropriately placed surface EMG electrodes reflect deep muscle activity (psoas, quadratus lumborum, abdominal wall) in the lumbar spine. journal of Biomechanics, 29 (11): 1 503-1 507. McGill, S.M., and Norman, RW. (1985) Dynamically and statically determined low back moments during lifting. journal of Biomechanics, 18 (12): 877-885. McGill, S.M., and Norman, R.W. (1987a) Effects of an anatomically detailed erector spinae model on L4/L5 disc compression and shear. journal of Biomechanics, 20 (6): 591-600. McGill, S.M., and Norman, RW. 0987b) An assessment of intra-abdominal pressure as a viable mechanism to reduce spinal compression. Ergonomics, 30 (11): 1 565-1588. McGill, S.M., and Norman, RW. (1988) The potential of lumbodorsal fascia forces to generate back extension moments during squat lifts. journal of Biomedical Engineering, 10: 312-318. McGill, S.M., Patt, N., and Norman, R.W. (1988) Measurement of the trunk musculature of active males using CT scan radiography: Implications for force and moment generating capacity about the L4/L5 joint. journal of Biomechanics, 21 (4): 329-341. McGill, S.M., Santaguida, L., and Stevens, J. (993) Measurement of the trunk musculature from T6 to L5 using MRI scans of 15 young males corrected for muscle fibre orientation. Clinical Biomechanics, 8: 171-178. McGill, S.M., Seguin,]., and Bennett, G. (994) Passive stiffness of the lumbar torso about the flexion-extension, lateral bend and axial twist axes: The effect of belt wearing and breath holding. Spine, 19 (6): 696-704. McGill, S.M., Thorstensson, A., and orman, RW. (1989) Non-rigid response of the trunk to dynamic axial loading: An evaluation of current modelling assumptions. Clinical Biomechanics, 4: 45-50. Santaguida, L., and McGill, S.M. (1995) The psoas major muscle: A three-dimensional mechanical modelling study with respect to the spine based on MRI measurement. journal of Biomechanics, 28 (3): 339-345. Sutarno, C, and McGill, S.M. (1995) Iso-velocity

investigation of the lengthening behaviour of the erector spinae muscles. Europeanjournal of Applied Physiology and Occupational Physiology, 70 (2): 146-1 53. THIS PAGE INTENTIONALLY LEFT BLANK Epidemiological Studies on Low Back Disorders (LBDs) Definitive experiments are rare in the fields of science and medicine. Instead, conclusions generally emerge from the integration and synthesis of evidence from a variety of sources. Using a similar approach, lawyers argue cases in which each piece of evidence is considered circumstantial in the hope that eventually the circumstantial evidence will become overwhelming. Like lawyers, scientific researchers gather and regather circumstantial evidence from several perspectives with the goal of understanding cause and effect. By studying the relationships among variables and investigating mechanisms, they are able to form perspectives that are robust and plausible. This type of research work, together with longitudinal studies, tests the causative factors identified in the mechanistic studies. Other chapters in this book are dedicated to investigating the various mechanisms within the low back and their links with good health and disability. This chapter focuses on the study of associations of variables through various epidemiological approaches. Some readers will find this a boring chapter-so my students inform me. You may choose to skip this chapter. But those wishing a fuller understanding of the challenges that lie in building a strong foundation for the very best injury prevention and rehabilitation programs are encouraged to read on. Several sections in this chapter will help you understand the risk factors for low back trouble-specifically, the changes in personal factors and whether they cause back troubles or are a consequence of having them. For the purpose of this review, purported disabling low back troubles and possibly related work intolerance will be referred to collectively as low back disorders (LBDs). Furthermore, the term personal factors can include anthropometric and fitness variables, as well as motor control ability, injury history, and so forth. Upon completion of this chapter you will appreciate the epidemiological perspective, have a more complete appreciation for the positions taken in this book to reduce the economic impact of low back disorders (LBDs), and understand certain subsequent approaches for prevention and rehabilitation. 29 30 Low Back Disorders Multidimensional Links Among Biomechanical, Psychosocial, and Personal Variables As noted in chapter 2, several prominent people have declared that psychosocial variables are the most significant factors in LBD. This is an important issue since effective intervention must address the real cause and consequence of back troubles. In this section we will see that virtually all studies that properly measured or calculated the phYSical demands of tasks found that people subjected to specific mechanical stressors are at higher risk of LBD but that there also appear to be some mitigating issues. Three Important Studies The following three studies are reviewed here because of their past influence and future implications. • Bigos et al. In 1 986 Bigos and colleagues performed a highly quoted study-one that has been very influential in shaping opinion regarding injLIly prevention and rehabilitation-at the Boeing plant in Washington State in the United States. This retrospective investigation analyzed 4645 injuries (of which 900 were to the low back) over a 1 5-month period in 1 979-1 980. They reported a correlation between the incidence of back injulY and p oor appraisal ratings of employees performed by their supervisors within six months prior to the reported injury. The authors considered the poor ratings to represent a psychosocial factor. In 1991 Bigos and colleagues conducted a longitudinal prospective study of 3020 employees at Boeing, during which there were 279 reported low back injuries. The researchers collected personality inventories as well as questionnaires regarding family and coworker support and job satisfaction. They also analyzed personal factors such as isometric strength, flexibility, aerobic capacity, height, and weight. The authors concluded that psychosocial

measures-particularly those related to job enjoyment-had the strongest influence of all the variables analyzed. In fact, those who stated they did not enjoy their job were 1.85 times more likely to report a back injury (odds ratio = 1 .85). Job satisfaction counted for less than 1 5% of the variance as an injury risk factor, meaning that more than 85% of the variance w as unaccounted for. In other words, psychosocial factors failed to account for 85% of the causation of LBDs. The authors concluded "that the statistically significant, though clinically modest, predictive power of work perceptions and psychosocial factors for reports of acute back pain among industrial workers argues against the exclusive use of an injury model to explain such problems." This is a fair summary of the implications of their work. It does not mean that mechanical loading is unimportant. Yet, this study is often quoted to support the viewpoint that psychosocial factors are the most important causes of back disorders. Interestingly, Marras and colleagues (993) also found similar odds ratios for job satisfaction 0.56) in a massive study of 400 repetitive industrial lifting jobs across 48 industries. Odds Ratios Perhaps the most l ucid definition of an odds ratio can be achieved through an example. If smokers have three times the risk of developing lung cancer as nonsmokers do (perhaps 6 out of 10 smokers as opposed to 2 out of 10 in nonsmokers), they have an odds ratio of 3. Thus, an odds ratio greater than 1 suggests an increased risk from a specific factor. Epidemiological Studies on Low Back Disorders (LBDs) 31 Very few epidemiologically based studies have employed reasonably robust quantifications of biomechanical, psychosocial, and personal factors. Two important studies meet this requirement. • Marras et al. The first important study was reported by Manas and colleagues (995), who surveyed over 400 industrial lifting jobs across 48 different industries. They examined medical records in these industries to classify each type of job as being either low, medium, or high risk for causing LBD. They documented a variety of mechanical variables as well as reported job satisfaction. The most powerful single variable for predicting those jobs with LBD was maximum low back moment (odds ratio = 4.04) between low- and medium-risk groups and which produced an odds ratio of 3.32 between the low- and high-risk groups. Other single variables produced impressive odds ratios, for example, sagittal trunk velOCity (odds ratio = 2 .48) for the low- and high-risk comparison and 2.42 for maximum weight handled between the low- and high-risk groups. Job satisfaction produced an odds ratio of 1.48 between the low- and high-risk groups and 1.32 between the low- and medium-risk groups. The researchers entered the single variables into a multiple logistic regression model. The group of various measures selected by the model described the risk index well between the low- and high-risk groups and also between the low- and medium-risk groups. Suitably varying the five measures chosen by the regression process (maximum load moment, maximum lateral trunk angular velocity, average trunk twisting velOCity, lifting frequency, and the maximum sagittal trunk angle) decreased the odds of being a member of the high-risk LBD group over 10 times (odds ratio = 10.6). This was an important study for specifying certain physical characteristics of job design that reduce the risk of LBD and linking epidemiological findings with quantitative biomechanica l analysis and psychosocial factors across a large working population. • Norman et al. The second important study to successfully integrate biomechanical, psychosocial, and personal factors was conducted by Norman and colleagues (998), who examined injuries that occurred in an auto assembly plant that employed more than 10,000 hourly paid workers. During a two-year period of observation in the plant, the authors reported analyses on 104 cases and 130 randomly selected controls. Cases were people who reported low back pain (LBP) to a nursing station; controls were people randomly selected from company rosters who did not report pain. This is a notable study because the authors attempted to obtain good-quality psychosocial, personal, and

psychophysical data on all participants from an interviewer-assisted questionnaire, as well as good-quality, directly measured biomechanical data on the physical demands of the jobs of all participants. (Note that the psychophysical approach is based on worker self-perceived stresses.) The study revealed that several independent and highly significant biomechanical, psychosocial, and psychophysical factors (identified as risk factors) existed in those who reported LBP. The personal risk factors that were included were much less important. After adjusting for personal risk factors, the statistically independent biomechanical risk factors that emerged were peak lumbar shear force (conservatively estimated odds ratio = 1 .7), lumbar disc compression integrated over the work shift (odds ratio = 2 .0), and peak force on the hands (odds ratio = 1.9). The odds ratios for the independent psychosocial risk factors, from among many studied, were worker perceptions of poorer workplace social environment (2.6), higher job satisfaction (not lower, as shown in the Boeing study) 0.7), higher coworker support 0.6), and perception of being more highly educated (2.2). Perceptions of higher physical exertion, a psychophysical factor, resulted in an odds ratio of 3.0, which is possibly related to the capacity of the worker relative to the job demands. Nearly 45% of the total variance was accounted for by these risk factors, with approximately 12% accounted for by the psychosocial factors and 31% by the 32 Low Back Disorders YOUR SACK WILL SE FINE LIFTING THESE 300 LB BOXES - I'LL GIVE YOU A GOOD EMPLOYEE REPORT! biomechanical factors. These results are very consistent with those reported by Marras and colleagues (1995) and by Punnett and colleagues (1991). Only a few of the personal factors were associated with reporting LBP: body mass index (odds ratio = 2.0) and prior compensation claim (odds ratio: 2.2). This case/control study is of high quality because it used a battery of many of the best measurement methods available for field use to assess many psychosocial, biomechanical, and personal factors on all participants in the data pool. The evidence from the comprehensive studies suggests that both psychosocial and biomechanical variables are important risk factors for LBD. In particular, cumu lative loading, joint moments, and spine shear forces are important. Those claiming that only psychosocial factors are i mportant or that only physical loading factors are important cannot mount a creditable, data-based defense, as it a ppears that the data they quote fail to measure properly either (or both) physical or psychosocial variables. Do Workers Experience LBDs Because They Are Paid to Act Disabled? Some papers in the literature appear to dismiss the link between pain and disability. Most of these papers clearly state that this notion is restricted to "nonspecific back pain," noting that specific diagnoses do impair the ability of a worker to perform a demanding job. However, some based their argument on the concept that low back tissue injury heals in 6 to 12 weeks, while others base their arguments on a behavioral model of chronic pain that is not totally consistent with the findings of other scientific approaches. A short discussion of the issues and evidence related to physician diagnosis, compensation, tissue damage, and pain is necessary. The position that chronic pain and disability are a function of compensation (and not mechanical factors) is contradicted by evidence that low back troubles continue after legal settlement of injury compensation (Mendelson, 1982). Hadler (1991) believes that the contest between the patient and the medical officer charged with determining Epidemiological Studies on Low Back Disorders (LBDs) 33 the compensatory award causes the patient to act disabled and thwarts any incentive to act well. Teasell and Shapiro (1998) shared this opinion in a review of several different chronic pain disorders, as did Rainville and colleagues (1997) in another very nice study that specifically addressed chronic low back pain. Unfoltunately, Hadler has taken the position that mechanical factors are, for the most part, of little importance in either causing or rehabilitating bad backs when compared with the psychosocial modulators (e.g.,

Hadler, 2001). His selective citation of the literature excludes evidence linking mechanical overload to tissue damage and ignores several important intervention studies. For example, Werneke and Halt (2001) showed that pain patterns upon patient presentation, specifically whether the pain "centralizes" or not, are much more powerful predictors of chroniCity than the psychosocial variables they studied. Thus, although the topic of compensation is important, it is irrelevant in discussions of the l inks between loading and LBD. Compensation issues should not be used to argue against the existence of a mechanical link between injury and work tolerance or, worse yet, to suggest that the removal of compensation will eliminate the cause of tissue damage. Does Pain Have an Organic Basis-Or Is It All in the Head? Much has been written about the apparent absence of an organic basis for chronic low back disability (and other chronic pain syndromes). As noted in chapter 1, as high as 85% of disabling LBD cases are claimed to have no definitive pathoanatomical diagnosis (White and Gordon, 1982). Two conclusions have been proposed: • Many LBD patients present with "nonorganic signs" suggesting psychological disturbance as the cause of their condition. • Poor diagnostic techniques, either from inadequately trained doctors or from limitations of widely available diagnostic technology, have precluded the making of many solid diagnoses. The first conclusion suggests that the correct course of action in these "undiagnosed" cases is to ignore physiological issues and address only psychological/psychosocial factors. The second conclusion suggests that one should not ignore the possibilities that more thorough diagnostic techniques could unearth physical causes and that rehabilitation based on that assumption could be more effective than rehabilitation for psychological disturbance alone. Let's examine each of these arguments. Does Absence Of DiagnOSiS Imply Psychological Cause? Waddell and colleagues wrote many manuscripts and guidelines (e.g., 1 980, 1984, 1987) on nonorganic signs in patients to support the notion of psychological disturbance overriding any pathoanatomical tissue damage (because none was diagnosed). Teasell and Shapiro (1998) wrote a nice summary of an extensive experimental literature suggesting that these pain symptoms may indeed have a physiological basis. They reviewed the recent science on the spread of neuron excitability and sensitization of adjacent neurons to explain the sensation of radiating pain in chronic conditions. Changes in neuroanatomy are coupled with biochemical changes with chronic pain. For example, in fibromyalgia patients numerous studies have shown levels of substance "P" in the cerebrospinal fluid elevated two to three times over controls (reported in a review by Teasell, 1 997). While the nonorganic signs described in Waddell's papers are important considerations in many cases and are a contribution to clinical practice, strong evidence suggests that many nonorganic signs may not be exclusive of a pathoanatomical mechanism that has eluded diagnosis. 34 Low Back Disorders Some have argued that no link exists between pain and tissue damage and activity intolerance. In the absence of no direct evidence, some groups have simply assumed that nociceptive pain that is not surgically correctable or that has not improved within six weeks should not be regarded as disabling (e.g., Fordyce, 1 995), In fact, the Fordyce monograph initiated much discussion, including several letters (e.g., Thompson/Merskey/Teasell/Fordyce, 1 996). Fordyce's 1 996 statements that "the course we are presently on threatens disaster" and that "we change or go broke" were particularly revealing. The high cost of treating chronic pain and disability appears to have motivated the elevation of the importance of the psychosocial factors so emphaSized in this monograph. It is also interesting to observe the absence of any eminent biomechanical expert among the author list of the Fordyce report. The Canadian Pain Society stated that the Fordyce report literature review "is incomplete and does not reflect the contemporary understanding of

chronic low back pain" (Pain, vol. 65, p. 114, 1996). Moreover, the Fordyce report largely ignored the evidence linking mechanical overload to measurable changes in spine biomechanics and spinal pain neuromechanical mechanisms. Another assumption of the report was that most spine LBDs are nonspecific, meaning simply that a diagnosis was not made. Could Inadequate Diagnosis Be a Fador in Nonorganic LBP? Bogduk and colleagues (996) argued that pain arising from many spinal tissues can be attributed to a detectable painful lesion. For example, the facet joints will produce pain upon stimulation (McCall, Park, and O'Brien, 1979). Bogduk's point is certainly correct. However, because not all lesions are easily detectable, it cannot be used to argue that if a lesion is not detected there is no organic basis for pain. For example, fractures and meniscal tears that have been detected in postmortem studies have not shown radiologically on planar X ray (Jonsson et al., 1 99 1; Taylor, Twomey, and Corker, 1990) or on CT (Schwarzer et al., 1 995). Nor have freshly produced fractures and articular damage been outwardly detectable radiographically in animal models (Yingling and McGill, 2000). Yet these are the typical diagnostic procedures used. Interestingly, Bogduk and colleagues (996) and Lord and colleagues (996) showed that injection of anesthetic (placebo-controlled diagnostic blocks) convincingly demonstrates that facet joints are often the site of pain origin. Further, disc studies examining the pain response of well over 1 000 discs in over 400 people undergoing discography by Vanharanta and colleagues (987) (subsequently reappraised by Moneta et al. in 1994 and reported by Bogduk et al. in 1996) showed a clear and statistically significant correlation between disc pain and grade 3 fissures of the annulus fibrosis. Most physicians would probably not detect these deep fissures of the annulus. In a rigorous and systematic study of diagnosis based on anesthetic blocks, Schwarzer and colleagues (995) were able to diagnose over 60% of the LBD cases cited in the study by Bogduk and colleagues (996) as being internal disc disruption (39%), facet jOint pain 0 5%), and sacroiliac pain 02%). Bogduk and colleagues (996) stated, "If inappropriate tests such as EMG and imaging are used, nothing will be found in the majority of cases, falsely justifying the impression that nothing can be found." Clearly we must question the statement that 85% of LBD cases are idiopathic or have no definitive pathoanatomical cause. Helpful Strategies for Undiagnosed LBDs Some physicians are clearly frustrated with the delayed improvement of undiagnosed chronic LBD patients. This frustration, together with concern for the financial health of the compensation system, may have motivated the Fordyce report to emphaSize psychosocial modulators rather than organically based variables to explain intolerance to certain types of activity. But dealing with frustration on the basis of false assumptions Epidemiological Studies on Low Back Disorders (LBDs) 35 will not help the situation. What, then, are more useful approaches to undiagnosed chronic cases of LBD? As noted earlier, many clinicians do not have the expertise or tools to diagnose back troubles at a tissue-based level. Provocative testing will enable many physicians to identify painful motions and loading. By integrating biomechanics with such testing, physicians may be aided in making functional diagnoses. In a small study Delitto and colleagues (995) suggested that appropriately classified back pain sufferers (those with functional classifications rather than tissue-specific diagnoses) do better with specific treatments. Further, as patients progress through the rehabilitation process, they seem to require different treatment approaches. For example, several studies suggest that manipulation can be beneficial for acute short-term troubles, while physical therapy and exercise approaches appear better for chronic conditions (Skargren, Carlsson, and Oberg, 1 998). A major limitation of these studies is ' that none has assessed progressive treatments that change as the patient progresses through the rehabilitation process; rather, they all have assessed only single treatment \ approaches. Future studies

must assess the efficacy of staged programs in which cat- \ egorized patients follow progressive treatment involving several sequenced approaches. In summaty, the position suggesting that there is no detectable pathoanatomical basis for pain and activity intolerance in some patients and thus that they are functions of only psychosocial variables does not appear to be defendable. I mproved tissue-based diagnosis, improved provocative/functional diagnosis, and better understanding of the interactions of psychological variables with pathoanatomical variables appear to have promise for helping to improve treatment outcomes. Are Biomechanical Variables and Psychosocial Variables Distinct? Are biomechanical variables and psychosocial variables distinct, or is there an interplay between them that, if understood, would underpin a more evidenced-based intervention program? Most repotts have made a clear separation between psychosocial and biomechanical factors. But is there any evidence that psychosocial factors could modulate musculoskeletal loading-or vice versa? First, consider that highly respected pain scientists present volumes of empirical data demonstrating that pain perception is modulated by sensory, neurophysiological, and psychological mechanisms, suggesting that the separation of the two for analysis is folly (e. g., Melzack and Wall, 1 983). Teasell (997) argued quite convincingly that, while psychological factors have been cited as being causative of pain and disability, in fact, psychological difficulties arise as the consequence of chronic pain (see also Gatchel, Polatin, and Mayer, 1 995; Radanov et aI., 1 994) and disappear upon its resolution (see also Wallis, Lord, Bogduk, 1997). The evidence is compelling-pain and psychological variables appear to be linked . Is there more direct evidence that psychosocial factors are inextricably linked with biomechanical factors? In a recent repolt, Marras and colleagues (2000) noted that certain personality factors, together with some psychosocial variables, appear to increase spinal loads by up to 27% in some personality types via muscular cocontraction. This appears to occur at moderate levels of loading, while biomechanical loading overrides any psychosocial effects under larger task demands. This conclusion was strengthened with a field study linking these general mechanistic observations with reported LBD in workers. In summary, LBDs appear to be associated with both loading and psychosocial factors, and these factors seem to be related and multifactorial. The same conclusion appears to be valid for many types of chronic pain conditions (Gamsa, 1 990). 36 Low Back Disorders What Is the Significance of First-Time Injury Data for Cause and Prevention? One of the best indicators of future back troubles is a previous histOlY of back troubles (Bigos et al., 1 991; Burton, Tillotson, Troup, 1 989; Troup et al., 1987). This suggests that studies of first-time back trouble episodes may be quite revealing for causative factors. BUlton, Tillotson, Symonds, et al. (1996) studied police officers in Northern Ireland wearing >8 kg (18 lb) of body armor in a jacket (this additional load was borne by the low back). This group demonstrated a shorter period of time to their first onset of pain when compared to officers in an English force that did not wear the body armor. The authors also found that spending more than two hours per day in a vehicle comprised a separate risk for first-time onset of LBP. Another survey (Troup, Maltin, and Lloyd, 1981) noted that falls among employees across a variety of industries were a common cause of first-time onset and were associated with longer periods of sick leave and a greater propensity for recurrence than were injuries caused by other mechanisms. (Ligamentous damage resulting from this type of loading is discussed in chapter 4.) It is also interesting to note that personal factors appear to play some role in first-time occurrence. Biering-Sorensen (1984) tested 449 men and 479 women for a variety of physical characteristics and showed that those with larger amounts of spine mobility and less lumbar extensor muscle endurance (independent factors) had an increased occurrence of first-time back troubles. Luoto and colleagues

(1995) reached similar conclusions. Muscular endurance, and not anthropometric variables, appears to be protective. Some injuries just happen as the result of motor control errors. (This interesting mechanism is introduced in chapter 4, where we describe witnessing an injll1Y using videoflouroscopy to view the spine.) These may be considered random events and may be more likely in people with poor motor control systems (Brereton and McGill, 1999). How Do Biomechanical Factors Affect LBO? Several approaches have provided evidence into the l inks between biomechanical factors and LBD. A few are summarized here. Mechanical Loading and LBO: Field-Based Risk Factors Of the epidemiological studies that have focused on kinematic and kinetic biomechanica l factors, a few investigated loading of low back anatomical structures. These would be considered to be the strongest evidence. The majority of the studies, however, assessed indirect measures that are linked to spinal loading such as the presence of static work postures, frequent torso bending and twisting, lifting demands, pushing and/or pulling exertions, and exertion repetition. While tissue overload is the cause of tissue damage and related back troubles, these indirect measures of load merely act as surrogates. The attraction of using surrogate measures rather than direct tissue loads per se for epidemiological study is that they are simpler to quantify and survey in the field. However, trade-offs exist among methodological utility, biological reality, and robustness. Risk factors related to specific tissue-based injury mechanisms are found in chapter 4. Several issues should be kept in mind when interpreting this l iterature. Virtually all reviews of the epidemiological literature (e.g., Pope, 1 989; Andersson, 1 991) have noted that specific job titles and types of work are associated with LBD (although LBD Epidemiological Studies on Low Back Disorders (LBDs) 37 is defined differently in different studies). In particular, jobs characterized by manual handling of materials , sitting in vibrating vehicles, and remaining sedentalY are all linked with LBD. However, this type of data does not reveal much about the links between specific characteristics of the work and the risk of suffering LBD; specifically, a dose-response relationship has not been elucidated. Furthermore , a review of 57 papers that surveyed low back disorders revealed no consistency between specific risk factors and the development of those disorders (Ferguson and Marras, 1 997). This review demonstrated the large differences in the way surveillance was performed and in risk factor measurements. We are reminded once again that epidemiological approaches alone will not elucidate the biological pathway of the development of LBDs , a process that must be understood to develop optimal prevention and rehabilitation strategies. As noted, the majority of specific risk factors that are addressed in the epidemiological literature (which is surprisingly sparse) are really surrogate factors, or indirect measures , of spine load. These surrogate factors are static work postures; seated work postures; frequent bending and twisting; lifting, pulling, and pushing; and vibration (especially seated). • Static work postures. Research has suggested that work characterized by static postures is an LBD risk factor. While many studies have suggested a link with static work, the key paper on this topic was presented by Punnett and coworkers (1991), who reviewed 1 995 back injury cases from an auto assembly plant. Analyzing jobs for postural and lifting requirements, they found that low back disorders were associated with postures that required maintaining mild trunk flexion (defined as the trunk flexed forward from 21 to 45°) (odds ratio = 4.9), postures involving maintaining severe trunk flexion (defined as the trunk being flexed forward greater than 45°) (odds ratio = 5.7), and postures involVing trunk twisting or lateral bending greater than 20° (odds ratio = 5.9). Their results suggested that the risk of back injury increased with exposure to these deviated postures and with increased duration of exposure. Deviated postures greatly increase low back tissue loading, particularly when they must be held (Marras, Lavender, and LeUl'gens et al., 1 993;

McGill , 1997). • Seated work postures. Kelsey (1975) linked the seated work posture to a greater risk of LBD. In a more recent study, Liira and colleagues (1996) suggested that although white collar (sedentalY) workers who must sit for long periods have a greater risk of low back troubles (8% increase in odds risk), active blue collar workers gain some prophylactic effect from sitting down (14% reduction in odds risk). This suggests that variable work, and not too much of any single activity, may have merit in reducing mechanically induced low back troubles. • Frequent bending and twisting. The U.S. Department of Labor report (1982) and many more studies (summarized by Andersson, 1 981; Marras et al., 1 995; Punnett et al., 1991; Snook, 1982) noted the increased risk of LBD from frequent bending and twisting. In fact, Marras documented in several studies the increased risk of LBD with higher torso velocities (e.g. , Marras et al., 1993, 1995). (Note that this is isolated spine motion and not nonspecific torso motion.) While these studies did not examine a mechanism to explain a link with LBD, the associated motion within the spine will be shown in chapter 4 to form a pathomechanism for very specific disabling low back disorders. • Lifting, pulling, and pushing. The National Institute for Occupational Safety and Health report (NIOSH , 1 981) provides a good review linking activities requiring lifting, pushing, and pulling with increased risk of LBD. • Vibration. Finally, vibration, particularly seated vibration, is linked to elevated rates of LBD (e.g., Kelsey, 1 975; Pope , 1 989). 38 Low Back Disorders While all of the risk factors noted here have been epidemiologically linked with an increased incidence of LBD, a subsequent section on tissue damage will provide insight into the mechanisms linking mechanical overload, the onset of pain and disability, and the natural history of these injuries as they pertain to job performance. What Are the Lasting Physiological, Biomechanical, and Motor Changes to Which Injury Leads? The following discussion of recent studies illustrates the substantial literature documenting specific performance deficits and subsequent anatomical changes in LBP populations. • Several studies have documented a change in muscular function after injuly (nicely summarized in Sterling, lull, and Wright, 2001). These include, for example, studies of the delayed onset of specific torso muscles during sudden events (Hodges and Richardson, 1 996, 1 999) that impair the spine's ability to achieve protective stability during situations such as slips and falls, changes in torso agonist/antagonist activity during gait (Arendt-Nielson et al., 1 995), and asymmetric muscle output during isokinetic torso extensor efforts (Grabiner, Koh, and Ghazawi, 1 992) that alters spine tissue loading. • Ariatomical changes following low back injury include asymmetric atrophy in the multifidus (Hides et al., 1 994) and fiber changes in the multifidus even five years after surgelY (Rantanen et al., 1 993). Further, in a very nice study of 108 patients with histories of chronic LBD ranging from 4 months to 20 years, Sihvonen and colleagues (1997) noted that 50% had disturbed joint motion and 75% of those with radiating pain had abnormal electromyograms to the medial spine extensor muscles. • Finally, a recent study of those with a history of low back troubles has shown a wide variety of l ingering deficits (McGill et al., in press). The back troubles were sufficient to cause work loss, but the subjects had been back to work for an average of 270 weeks. Generally, those with a history of troubles were heavier and had disturbances in the flexor/extensor strength ratio and the flexor/extensor endurance ratio together with the lateral bend endurance ratio, had diminished gross flexion range of lumbar motion, and had lingering motor difficulties compromising their ability to balance and bend down to pick up a light object. None of these changes could be considered to be good. The broad implication of this work is that a histolY of low back trouble, even when a substantial amount of time has elapsed since the trouble, is associated with a variety of lingering deficits that would require a multidisciplinalY intervention approach to diminish their presence. This collection of

evidence is quite powerful in documenting pathoneuromechanical changes associated with chronic LBD. These changes are lasting years-not 6 to 12 weeks! What Is the Optimal Amount of Loading for a Healthy Spine? Lucid interpretation of the data in the epidemiological literature is limited by the fact that the levels at which tissue damage occurs remain obscure. Many are concerned with the known tissue damage that occurs with high magnitudes of load, repetition, and so on. For example, Herrin and colleagues (1986) found that musculoskeletal injuries were twice as likely if the worker'S lumbar spine was exposed to compressive forces that exceeded 6800 newtons (predicted with a biomechanical model). On the other hand, several of the epidemiological studies have not been able to support a Epidemiological Studies on Low Back Disorders (LBDs) 39 link between heavy work, when crudely measured, and the risk of LBD (e.g. , Bigos et aI., 1 986; Porter, 1987). Mitigating factors appear to include repetition of similar movements and variety in work. In the discussion of the U-shaped relationship between activity levels and LBD discussed in chapter 1, we saw that stress at optimal levels strengthens the system, while too little or too much is detrimental to health. Kelsey (1975) demonstrated a greater than expected increase in disc protrusions among sedentalY workers. While many consider sitting a "low load" task, in fact it creates damaging conditions for the disc-the mechanism of which will be explained in chapter 4. This has obscured the emergence of a much clearer relationship among biomechanical loading, disc herniation, clinical impressions, and the ability to perform demanding work. Futther, Videman and colleagues (1990) studied a cross-section of retired workers by comparing their LBD history with their MRI scans. A history of back pain and the visible parameters of spinal pathology were least prevalent in workers whose jobs had included moderate activity and most prevalent in workers with either sedentary or heavy work. Many studies compare just two levels of work-for example, light versus heavy, light versus moderate, or moderate versus heavy. Because any relationship other than a straight line requires more than two points (or levels of activity or levels of loading), such studies cannot illustrate the U-shaped relationship. Consider the data compiled in a thorough epidemiological study reported by Liira and colleagues (1996) of LBD prevalence and physical work exposures. Even though the authors considered only the upper levels of excessive loading, they concluded that one quarter of excess back pain morbidity could be explained by physical work exposures. Real possibility remains that this is an underestimate. Furthermore, the nature of LBD appears to be affected by the type of work. Videman and colleagues (1990) noted a tendency among those who had had sedentary careers to have marked disc degeneration in later years, while those who had performed heavy work (defined as not only lifting but also requiring large trunk motions) tended to have classic arthritic changes in the spine (stenosis, osteophytosis, etc.). In a similar study, Battie and colleagues (1995) reported an apparent contribution of genetic factors to various age-related changes in the spine using monozygotic twins, given significantly greater similarities in spinal changes than would be expected by chance. Porter (1987, 1 992) performed two studies furthering the notion of an optimal loading level for health. The first study tracked miners and nonminers treated at hospitals for back pain. While Significantly more miners reported for back trouble compared with nonminers, Significantly fewer were diagnosed with disc protrusions, while Significantly more were reported to have stenosis and nerve root entrapment (conditions associated more with the arthritic spine according to the data of Videman, Nurminen, and Troup, 1 990). The second study evaluated questionnaire results from 1 96 patients with symptomatic disc protrusion and 53 with root entrapment syndrome. They were asked about their histolY of heavy work between the ages of 15 and 20 years. Significantly more subjects with disc protrusion had done no heavy physical work

in those early years. By contrast, more of those with nerve entrapment syndrome had done five full years of heavy work between 15 and 20 years of age. This collective data suggests that different work demands cause different spine conditions and perhaps that the optimum loading is different for different tissues. Nonetheless, the optimum activity appears to be varied work at a moderate level between sedentary and heavy work. What constitutes an optimum load-a load that is not too much, not too little, not too repetitive, and not too prolonged? Currently, we are in need of assessment tools for determining optimum load to guide the intervention toward optimum health. 40 Low Back Disorders What Are the Links Between Personal Factors and LBO? Some personal factors such as muscle endurance (not strength) and less spine range of motion (not more) are prophylactic for future back troubles. These will be addressed in part III, Low Back Rehabilitation. A few other personal factors are noted here. A few specific personal factors appear to affect spine tissue tolerance according to the existing literature; age and gender are two examples. Jager and colleagues (1991) compiled the available literature on the tolerance of lumbar motion units to bear compressive load that passed their inclusion criteria. Their results revealed that when males and females were matched for age, females were able to sustain only approximately two thirds of the compressive loads of males. Furthermore, Jager and colleagues' data showed that within a given gender, the 60-year-old spine was able to tolerate only about two thirds of the load tolerated by the 20-year-old spine. Keep in mind that age and gender are very simple factors. It appears that other personal factors, such as poor motor control system "fitness," can lead to a back injLllY during ordinarily benign tasks such as picking up a pencil from the floor. The modeling data of Cholewicki and McGill (1996) suggest that the spine can easily buckle during such a task. When the muscle forces are inherently low, a small motor error can cause rotation of a single spinal joint, placing all bending moment support responsibility on the passive tissues. Such scenarios do not constitute excessive tasks, but patients often report them to clinicians as the event that caused their injury. This phenomenon will not be found in the scientific literature, however. Many medical personnel would not record this event as the cause of injury since in many jurisdictions it would not be deemed a compensable injury. These types of injuries seem to be more influenced by the fitness of an individual's motor control system than by factors such as strength. McGill and colleagues (1995) noted that people differ in their ability to hold a load in their hands and breathe heavily. This is very significant since the muscles required to be continuously active to SUppOlt the spine (and prevent buckling) are also used to breathe by rhythmically contracting. Those who must use their muscles to breathe in this way sacrifice spine stability. This is particularly noticeable in those with compromised lung elasticity (smokers, emphysemics, etc.). All of these deficient motor control mechanisms will heighte biomechanical susceptibility to injury or reinjury (Cholewicki and McGill, 1996) and are highly variable personal characteristics. Additional factors other than simple load magnitude appear to modulate the risk of tissue damage. The mechanism of disc herniation provides an example. While disc herniations have been produced under controlled conditions (e.g. , Gordon et al., 1 991), they have not been produced conSistently. Our lab has been able to consistently produce disc herniations by mimicking spine motion and load patterns seen in workers (Callaghan and McGill, 2001). Specifically, it appears that only a very modest amount of spine compression force is required (only 800- 1 000 N), but the spine specimen must be repeatedly flexed-mimicking repeated torso-spine flexion from continual bending to a fully flexed posture. The main relevance for this issue is that the way in which workers elect to move and bend will influence the risk of disc herniation. This highlights the need to examine how workers move in standardized tests at the beginning of their careers while they still have "virgin" backs,

making it possible to determine cause and effect. Recent evidence suggests that those with a history of back troubles are more likely to lift flexing the spine and not the hips, increasing the risk of future back damage (McGill et al., in press). Epidemiological Studies on Low Back Disorders (LBDs) 41 What the Evidence Supports In summary, it is interesting to consider why only some workers become patients. There is no question that damage to tissue can be caused by excessive loading, and damage causes pain. However, pain is a perception that is modulated by psychosocial variables in addition to physiological injury. Clearly both psychosocial and biomechanical variables are associated with LBD and a re important in preventing low back injury and the ensuing chronicity; collectively the evidence from several scientific perspectives is overwhelming. The relative importance of either is often difficult to compare across studies as the metrics for each a re different-biomechanical variables are reported in newtons, newton-meters, x cycles, and so forth, while psychosocial variables are reported in ordinal scales lin ked to perception (independent risk factors can be compared using odds ratios). Some influential reports have ignored biomechanical evidence and promoted psychosocial variables as being more important. However, no study of psychosocial variables has been able to conclusively establish causal links-only association. Some biomechanically based studies, together with the chronic pain literature, are strongly convincing in their establishment of both association and causality. Thank goodness! We can now p roceed. Back injury can begin with damage to one tissue, but this changes the biomechanical function of the joint. Tissue stresses change and other tissues become involved leading to progressive deterioration with time. Tissue damage does not always result from too high a load magnitude. In the case of disc herniation, repetitive motion, even in the absence of large loads, seems to be a significant causative mechanism. The notion that tissues heal within 6 to 12 weeks and that longer-lasting work intolerance has no pathoanatomical basis appears to be false; tissue injUly data and the science of chronic pain mechanisms strongly suggest otherwise. Understanding the role of biomechanical, psychosocial, and personal factors together with their interrelationships will build the foundation for better prevention in the future. The challenge is to develop variable tolerance guidelines, psychosocial guidelines, and higher-level medical practice codes. On balance the evidence supports the following statements: • Biomechanical factors are linked to both the incidence of first-time low back troubles and absenteeism, and subsequent episodes. • Psychosocial factors appear to be important as well but may be more related to episodes after the initial back-related episode. • Psychosocial and biomechanical factors appear to influence each other, both in terms of causation of work absence and in the course of recovery. • The relationship between loading and LBDs appears to be a U-shaped function with the optimal loading being at a moderate level. • Low back tissue damage can initiate a cascade of changes that may cause pain and intolerance to certain activities, and these changes may be disruptive for up to 10 years in an unfortunate few. • Many types of tissue damage can escape detection in vivo. Even gross damage visible during dissection is often not visible on medical images. Thus, nonspecific diagnOSiS does not rule out the presence of mechanical damage and must not be used to imply that mechanical factors are not related to LBD. 42 Low Back Disorders References Andersson, G.B. (981) Epidemiologic aspects of low back pain in industry. Spine, 6: 53-60. Andersson, G.B. (1991) The epidemiology of spinal disorders. In:].W. Flymoyer (Ed.), The adult spine: Principles and practice (chapter 8). New York: Raven Press. Arendt-Nielson, L., Graven-Neilson, T, Svarrer, H., and Svensson, P. (1995) The influence of low back pain on muscle activity and coordination during gait. Pain, 64: 231-240. Battie, M.C, Haynor, D.R., Fisher, L.D., Gill, K, Gibbons, L.E., and Videman, T (1995) Similarities in degenerative findings on magnetic resonance images of

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five lumbar vertebrae. The construction of the vertebral bodies themselves may be likened to a barrel with round walls made of relatively stiff cortical bone (see figure 4.1). The top and bottom of the barrel are made of a more deformable cartilage plate (end plate) that is approximately .6 mm (.002 in.) thick but thinnest in the central region (Roberts, Menage, and Urban, 1989). The end plate is porous for the transport of nutrients such as oxygen and glucose, while the inside of the barrel is filled with cancellous bone. The trabecular arrangement within the cancellous bone is aligned with the stress trajectories that develop during activity. Three orientations dominate-one vertical and two oblique (Gallois and]apoit, 1925) (see figure 4.2, a-b). 45 Pedicle Spinous Transverse process Lamina _____ Superior articular process • Transverse process Transverse process Inferior articular facet Inferior articular process -:WlllL••--- Accessory Vertebral foramen ----•..;..,..:.....:..• Superior articular facet Transverse process • Lamina ----------,1 Inferior articular facet ------4-1 Figure 4.1 The parts of a typical lumbar vertebra. Image courtesy of Primal Pictures. Superior articular process Mamillary process Accessory process 1IiIIli .ti'-:;-------__ Spinous process c Figure 4.2 The arrangement of the trabeculae (first noted by Gallois and Japoit in 1925) is aligned with the dominant trajectories of stress. The three trabecular systems are shown in G, b, and c. 46 Functional Anatomy of the Lumbar Spine 47 Vertebral Architecture and Load Bearing The very special architecture of the vertebral bodies determines how they bear compressive load and fail under excessive loading. The walls of the vertebrae (or sides of the barrel) remain rigid upon compression, but the nucleus of the disc pressurizes (the classic work is by Nachemson, 1960, 1966) and causes the cartilaginous end plates of the vertebrae to bulge inward, seemingly to compress the cancellous bone (Brinckmann, Biggemann, and Hilweg, 1989). In fact, under compression the cancellous bone fails first (Gunning, Callaghan, and McGill, 2001), making it the determinant of failure tolerance of the spine (at least when the spine is not positioned at the end range of motion) . It is difficult to injure the disc annulus this way (annular failure will be discussed later) . Althought this notion is contralY to the concept that the vertebral bodies are rigid, the functional interpretation of this anatomy suggests a very clever shock-absorbing and load-bearing system. Farfan (973) proposed the notion that the vertebral bodies act as shock absorbers of the spine, although he based this more on vertebral body fluid flow than on end-plate bulging. He suggested that the discs were not the major shock absorbers of the spine, contrary to virtually any textbook on the subject! Since the nucleus is an incompressible fluid, bulging end plates suggest fluid expulsion from the vertebral bodies, specifically blood through the perivertebral sinuses (Roaf, 1960). This mechanism suggests a protective dissipation u pon quasistatic and dynamic compressive loading of the spine. More vertebral body-based shock-absorbing mechanisms are documented subsequently. In summary, the common statement found in many textbooks that the discs are the shock absorbers of the spine now appears questionable; rather, the vertebral bodies appear to play a dominant role in performing this function. Understanding Vertebral Mechanics To truly appreciate vertebral behavior I encourage you to obtain a vertebra from a butcher (bovine or porcine is ideal). Hold the vertebra end plate to end plate between your thumb and finger and squeeze. If you have never done this before, you will be amazed by the deformation and elasticity. The vertebra experiences similar deformation as the incompressible nucleus of the disc presses over the central end plate during spine compression in vivo. Deformable vertebrae is a new notion for many. How do the end plates bulge inward into seemingly rigid bone? The answer appears to be in the architecture of the cancellous bone. Vertebral cancellous bone structure is dominated by a system of columns of bone (shown in figure 4.2) that run vertically from end plate to end plate. The vertical columns are tied together with smaller transverse

trabeculae. Upon axial compression, as the end plates bulge into the vertebral bodies, these columns experience compression and appear to bend. Under excessive compressive load, the bending columns will buckle as the smaller bony transverse trabeculae fracture, as documented by Fyhrie and Schaffler (994) (see figure 4.3). In this way, the cancellous bone can rebound back to its original shape (at least 95% of the original unloaded shape) when the load is removed, even after suffering fracture and delamination of the transverse (trabecu lae). This architecture appears to afford excellent elastic deformation, even after marked damage, and then to regain its original structure and function as it heals. Damaged cancellolls fractures appear to heal quickly, given the small amollnt of osteogenic activity needed, at least when compared with the length of time needed to repair collagenous tissues. 48 Low Back Disorders Microfracturing of the trabeculae can occur with repetitive loading at levels well below the failure level from a single cycle of load. Lu and colleagues (2001) demonstrated that cyclic loading at 10% of u ltimate failure load caused no damage or change in stiffness, but with 20,000 cycles of load at 20-30% of the ultimate failure load, both stiffness and energy absorbed at failure were decreased. Highly repetitive loads, even at quite low magnitudes, a ppea r to ca u se microdamage. Figure 4.3 Under compressive loading, bulging of the end plate causes buckling stresses in the vertical trabeculae, which, when excessive, causes damage in the transverse trabeculae. Note the vertical (from compression) (A) and horizontal (from tension) (B) cracks in the transverse trabeculae. Reprinted from Bone, Vol. 1 5(1), Fyhrie and Scheffler, "Failure mechanisms in human vertebral cancellous bone," 1 05-1 09, Copyright 1 994, with permission of Elsevier Science. The osteoporotic vertebra is characterized by mineral loss and declining bone density in the trabeculae. Because transverse trabeculae are far fewer in number than longitudinal trabeculae and because they are generally of smaller diameter, the transverse trabeculae specifically are the target for mechanical compromise with osteoporotic mineral loss (Silva and Gibson, 1997) (see figure 4.4). Interestingly, the same authors noted a higher tendency for the transverse trabeculae to disappear in females with greater incia b dence than in males. This loss in mechanical integrity of the transverse trabeculae has a great influence on the compressive strength of the vettebrae via the mechanism described earlier. Thus, the osteoporotic vertebra begins to slowly collapse when exposed to excessive load, with serial buckling of the columns of bone ultimately developing the classic wedge shape. Figure 4.4 With aging, the transverse or horizontal trabeculae thin and eventually lose their ability to support the vertical trabeculae, which can then buckle, causing vertebral collapse. (a) A healthy trabecular bone network from a 47-year-old woman. (b) Perforation in a horizontal trabecula in an elderly woman. It is interesting to contrast the other extreme of the bone density spectrum. The transverse trabeculae harvested from specimens who performed heavy work (in particular, weightlifters) were thick and dense. In addition, where the transverse trabeculae intersected with the vertical columns, the Reprinted from Bone, Vol. 21, Silva and Gibson,"Modeling the mechanical behaviour of vertebral trabecular bone: Effects of age-related changes in microstructure," 1 91- 1 99, Copyright 1 997, with permission from Elsevier Science. joints were characterized with heavy bony gusseting, similar to what a welder would weld to strengthen a right-angled joint. The transverse trabeculae appear to be crucial in determining compressive strength. Yet another observed failure mechanism of the vertebral body is termed the "slow crush," in which extensive trabecular damage is observed without concomitant loss of stiffness or abrupt change in the load-deformation relationship (Gunning, Callaghan, and McGill, 2001) (see figure 4.5). Since slope change in the load-deformation relationship is often used to identify the yield point, or the initial tissue damage, this injury can go unnoticed. Interestingly enough, vertebrae failing

under ever-increasing compressive load will gradually increase in stiffness, a testament to the wonderful architecture of the transverse and vertical trabeculae. Also interesting is the load-rate dependence of bone strength, as the end plate appears to fail first at low load rates while the vertebral bony elements fail at higher load rates (see figure 4.6). a b Functional Anatomy of the Lumbar Spine 49 Figure 4.5 Massive trabecular damage found during the d issection and removal of marrow following an excessive "slow crush" compressive load (a). Higher magnification of the crush fracture is shown in (b). Even this massive fracture was not clear on X ray or any other examination methods. Reprinted with permission of J. Gunning, J. Callaghan, and S. McGill, Clinical Biomechanics, 1 6(6), 2001. Copyright by Elsevier Science. 100 .-.---.---------- en 60' Figure 5.2 The lumbo-pelvic rhythm is the textbook description of how people bend over. The first 60° takes place in the lumbar spine (flexing), while further rotation of the torso is accomplished with rotation about the hip. We have never measured this sequence in anyone-from professional athlete to back patient. (continued) 92 Low Back Disorders clinical textbooks, we have never measured this strict sequence in anyone. In fact, Olympic weightlifters attempt to do the opposite-they lock the lumbar spine close to the neutral position and rotate almost entirely about the hips. When the lumbar spine and hip interplay is quantified in most people, it is apparent that torso flexion is accomplished with a combination of hip motion and lumbar spine flexion. In fact, given the ligament and annulus stresses associated with lumbar flexion, avoidance of full spine flexion is both prophylactic and therapeutic for most patients. The belief that the lumbo-pelvic rhythm (with distinct separation of spine and pelvic motion) is beneficial, as described in so many textbooks, appears to be a clinical myth and not the product of quantified spine and pelvis motion. Kinetics and Normal Lumbar Spine Mechanics Interpretation of the function of the anatomical components of the lumbar spine requires • analysis of their architecture and neural activation of muscles, and • knowledge of forces in the individual tissues (both active and passive) during a wide variety of tasks. This information is crucial for understanding how tissue overloading and injuries occur and also for optimizing treatment strategies for specific spine injUly. Table 5.4 and figure 5.3 provide activation levels quantified with surface and intramuscular EMG for a variety of torso m uscles and over a variety of activi ties. These will be referred to throughout this book. This section will a ddress several issues and controversies about the functional interpre tation of the thoraco-lumbar anatomy. G iven the inability of the clinician and scientist to measure individual tissue forces in v ivo, the only tenable option is to use sophisticated measurement techniques to collect various biological signals from living subjects and integrate them with sophis ticated modeling approaches to estimate tissue loads. In chap ter 2, I described briefly the technique we used to assess the various issues in this chapter (the virtual spine). Standing and Bending Forward Several studies over the years have shown the flexion/relaxation phenomenon, or the apparent myoelectric silence of the low back extensor muscles during a standing-tofull-flexion maneuver. The hypothesis has been that, as full flexion occurs, either the extensors shut down their neural drive by reflex or the passive tissues simply take the load as they strain under full flexion. A study by McGill and Kippers (994) using the virtual spine approach described in chapter 2 quantified individual tissue forces, thus adding more insight into the understanding of this task. As one bends fOlward, the spine flexes and the extensors undergo eccentric contraction. As full flexion is approached, the passive tissues rapidly take over moment production, relieving the muscles of this role and accounting for their myoelectric silence. Figure 5.4 shows the relative contribution of the muscles and the passive tissues (ligaments, disc, and gut) to the reaction moment throughout the movement, while table 5.5 documents the distribution of tissue

forces and their moments and joint load consequence. Interestingly, the "relaxation" of the lumbar extensor muscles appeared to occur only in an electrical sense because they generated substantial force elastically during full spine flexion through stretching. Perhaps the term .flexion/relaxation is inappropriate, \0 W Table 5.4 Subject Averages of EMG Activation Normalized to That Activity Observed During a Maximal Effort (1 OO%)-Mean and (Standard Deviation in Parentheses) Psoas channels, external obl ique, i nternal oblique, and tra nsverse abdominis are i ntra muscular electrodes while rectus abdominis, rectus femoris, and erector spinae a re surface electrodes. Task Psoas 1 Psoas 2 EOi IOi TAi RAs RFs ESs Straight-leg sit-up 15(±12) 24(±7) 44(±9) 15(±15) 11 (±9) 48(±18) 16(±10) 4(±3) Bent-knee sit-up 17(±10) 28(±7) 43("12) 16(±14) 10(±7) 55(±16) 14(±7) 6(±9) Press-heel sit-up 28(±23) 34(±18) 51(±14) 22(±14) 20(±13) 51 (±20) 15(±12) 4(±3) Bent-kn ee curl-up 7(±8) 10(±14) 19(±14) 14(±10) 12(±9) 62(±22) 8(±12) 6(±10) Bent-knee l eg raise 24(±15) 25(±8) 22(±7) 8(±9) 7(±6) 32(±20) 8(±5) 6(±8) Straight-leg raise 35(±20) 33(±8) 26(±9) 9(±8) 6(±4) 37(±24) 23(±12) 7(±11) Isom. hand-to-kn ee (left hand ---> rig ht knee, 16(±16) 16(±8) 68(±14) 30(±28) 28(±19) 69(±18) 8(±7) 6(±4) right hand ---> l eft knee) 56(±28) 58(±16) 53(±12) 48(±23) 44(±18) 74(±25) 42(±29) 5(±4) Cross cu rl-up (right should er ---> across, 5(±3) 4(±4) 23(±20) 24(±14) 20(±11) 57(±22) 10(±19) 5(±8) l eft shoulder ---> across) 5(±3) 5(±5) 24(±17) 21(±16) 15(±13) 58(±24) 12(±24) 5(±8) Isom. side support (left side down) 21 (±17) 12(±8) 43(±13) 36(±29) 39(±24) 22(±13) 11 (± 11) 24(±15) Dyn. side support (left side down) 26(±18) 13(±5) 44(±16) 42(±24) 44(±33) 41 (±20) 9(±7) 29(±17) Push-up from feet 24(±19) 12(±5) 29(±12) 10(±14) 9(±9) 29(±10) 10(±7) 3(±4) Push-up from knees 14(±11) 10(±7) 19(±10) 7(±9) 8(±8) 19(± 11) 5(±3) 3(±4) Li ft light load (20 kg) 9(±10) 3(±4) 3(±3) 6(±7) 6(±5) 14(±21) 6(±5) 37(±13) Li ft h eavy load (50-110 kg) 16(±18) 5(±6) 5(±4) 10(±11) 10(±9) 17(±23) 6(±5) 62(±12) (continued) 1.O +::0 Table 5.4 (continued) l Task Psoas 1 Psoas 2 EOi IOi TAi RAs RFs ESs Symmetric bucket hold, 20 kg (441b) 2{±4) 1 (±1) 7{±4) 5(±3) 5(±1) 10(±7) 3{±3) 3{±6) 30 kg (661b) 3{±4) 1 (±1) 9(±5) 6{±4) 6{±1) 10(±8) 3{±3) 4{±7) 40 kg (881b) 3(±5) 1 (±1) 10(±6) 8{±6) 6{±2) 10(±8) 3{±3) 3{±2) o kg 1 (±2) O(±l) 2{±1) 2{±2) 2{±1) 10(±9) 2(±1) 2(±1) Seated isom. twist CCW 30(±20) 17(±15) 18(±8) 43(±25) 49(±35) 17(±22) 7{±4) 14(±6) Seated isom. twist CW 23(±20) 11 (±8) 52(±13) 15(±11) 18(±19) 13(±1O) 9(±10) 13(±8) Standing hip i nternal rotation 21(±18) 10(±9) 18(±12) 24{±23) 33(±20) 13(±9) 9{±7) 18(±6) Standing hip external rotation 27(±20) 22(±19) 17(±13) 21(±19) 31(±17) 13(±8) 19(±11) 17(±9) Sitting hip i nternal rotation 19(±15) 21(±18) 36(±31) 30(±30) 31 (±29) 18(±8) 20(±19) 12(±8) Sitting hip external rotation 32(±25) 25(±20) 11 (±9) 15(±17) 16(±13) 15(±9) 16(±13) 8{±8) Sitting u pright 12(±7) 7(±5) 3{±6) 3{±3) 4{±2) 17(±9) 4{±2) 5(±8) Sitting slouched /relaxed 4{±4) 3{±3) 2(±5) 2{±2) 4{±3) 17(±11) 3{±2) 5(±8) Qui et standing 2(±1) 1 (±1) 3{±4) 5(±3) 4{±2) 5(±5) 3{±3) 11(±11) Standing extended 3{±2) 2(±1) 12(±9) 6{±3) 5(±3) 11 (±5) 4{±3) 7{±8) Standing lateral bend, l eft 9(±10) 1 (±2) 11 (±8) 18(±14) 12(±7) 13(±7) 3{±2) 11(±13) --Standing lateral bend, right 6(±5) 1 (±2) 19(±18) 18(±14) l 25(±20) 14(±9) 3{±1) 8{±8) l Seated lateral bend moving l eft 2{±3) 1 (±1) 21(±19) 7{±7) 7(±11) 13(±8) 4{±3) 6{±7) Seated lateral bend, moving right 18(±12) 12(±2) 15(±26) 10(±7) 12(±7) l 17(±20) 5(±4) 5(±8) -Upright - 14(±9) 8{±4) 6{±4) 5(±3) 5(±5) 19(±23) 5(±3) 6{±8) ---- -- -----' l l Reprinted, by permission, from D.Juker, S.M. McGill, S.M. Kropf,T.Steffen, 1998,"Quantitative intramuscular myoelectric activity of lumbar portions of psoas and the abdominal wall during a wide variety of tasks," Medicine and Science in Sports and Exercise 30(2): 301-31 O. ©Lippincott, Williams, and Wilkins. Straight-leg sit-ups L Quiet standing ft t Bent-knee sit-ups " , Standing extended t Press-heel sit-ups " Standing lateral bendleft Bent-knee curl-ups Standing lateral bend- " right Bent-knee leg raise Seated lateral bend- moving left J: Straight-leg raise Seated lateral

bend- , moving right n Isometric hand-to-knee ◆ (I hand-r knee/r hand-I knee) Seated upright ◆ Cross curl-up ◆ (to r/I shoulder) Seated isometric twist (CW/CCW) Isometric side support ◆ t (left side down) Standing hip rotation (internal/external) ◆ Dynamic side support I (left side down) Seated hip rotation (internal/external) , , Push-up from feet ~ Sitting upright ~ ◆\ ◆ Push-up from knees Sitting slouched, relaxed Symetric bucket hold Lift light load (20 kg) ◆ \ (20/30/40/0 kg) Lift heavy load (50-1 00 kg) Figure 5.3 Schematic documenting various tasks during which EMG signals were obtained. They are listed in table 5.4. Reprinted, by permission, from D.Juker, S.M. McGill, S.M. Kropf, T. Steffen, 1998,"Quantitative intramuscular myoelectric activity of lumbar portions of psoas and the abdominal wall during a wide variety of tasks," Medicine and Science in Sports and Exercise 30(2): 301- 310. © Lippincott, Williams, and Wilkins. 95 E ◆ C Q) E 0 :2 96 Low Back Disorders 200 1 50 1 00 /', ◆/ '\ . . . . '\ / \ ,/ lj 50 \ V , \ . : , I: , '-- "-""""",,-- --' ... ---..... ' . " .. .._-------. .,/ ... , o +----.:;-;.;....o..a.:...;:..: .• :..: • ..:. • .:..... •• -.--r---------- -------- --'-◆.;.:. :... ..;;;;..;:;:;... -- o 3 6 Time (s) 9 12 Raw moment DiscTq LigTq _.- MuscleTq GutTq Figure 5.4 During standing to full forward flexion the extensor muscles eccentrically contract but transfer their moment supporting role (MuscleTq) to the passive tissues at full flexion-the disc (DiscTq), the buckled gut (GutTq), and the ligaments (LigTq). Note that some force remains in the muscles with passive stretching. particularly for those who may be attempting to minimize forces in the muscle in clinical settings. FUlthermore, the shear loading is substantial (see chapter 4 for a discussion of the ligament muscle vector directions and the loss of shear support in the extensors with full flexion) and would suggest caution for those with spondylolisthesis or other more subtle shear instabilities. Clearly, straight-leg toe touches or kneesto-chest stretches would cause similar concern. Loads on the Low Back During Lifting During lifting, muscle and ligament forces requi red to support the posture and facilitate movement impose mammoth loads on the spine. This is why lifting technique is so important to reduce low back moment demands and the risk of excessive loading. The following example demonstrates this concept. A man is lifting 27 kg (59 lb) held in the hands using a squat lift style. This produces an extensor reaction moment in the low back of 450 Nm 032 ft lb). The forces in the various tissues that support this moment impose a compressive load on the lumbar spine of over 7000 N (1 568 lb). Table 5.6 details the contributions to the total extension moment and to the forces from the muscular components. These forces and their effects are predicted using the sophisticated modeling approach, which uses biological Signals obtained directly from the subject (see chapter 2). It should be noted here that 7000 N (1568 lb) of compression begins to cause damage in very weak spines, although the tolerance of the lumbar spine in an average healthy young man probably approaches 1 2-15 kN (2688-3360 lb) (Adams and Dolan, 1 995). In extreme cases, compressive loads on the spines of competi tive weightlifters have safely exceeded 20 kN (4480 lb) (Cholewicki, McGill, and Norman, 1991). Understanding the individual muscle forces, their contribution to supporting the low back, and their components of compression and shear force that are imposed on the spine is very useful. In this palticular example, the lifter avoided full spine flexion Table 5.5 Individual Muscle and Passive Tissue Forces and Moments During Full Flexion The moment was 171 Nm (38 Nm by muscle, 113 Nm by ligaments, and 20 Nm by passive tissues, such as the disc, skin, and buckled viscera). The joint compression was 3145 N, and shear was 1026 N. Force Moment (Nm) Compression Shear (N) N Flexion Lateral Twist N Anteroposterior Lateral Muscle R rectus abdominis 16 -2 1 1 15 5 -4 L rectus abdominis 16 -2 -1 -1 15 5 4 R external oblique 1 , 10 -1 1 1 8 7 -3 L external oblique 1 10 -1 -1 -1 8 7 3 R external oblique 2 7 -1 1 0 6 2 -3 L external oblique 2 7 -1 -1 -0 6 2 3 R internal oblique 1 35 0 3 -2 21 -19 20 L internal oblique

1 35 0 -3 2 21 -19 -20 R internal oblique 2 29 -2 2 -3 8 -17 21 L internal oblique 2 29 -2 -2 3 8 -17 -21 R pars lumborum (L 1) 21 2 1 0 21 6 2 L pars lumborum (L 1) 21 2 -1 -0 21 6 -2 R pars lumborum (L2) 27 2 1 0 26 8 2 L pars lumborum (L2) 27 2 -1 -0 26 8 -2 R pars lumborum (L3) 31 1 1 0 29 -4 6 L pars lumborum (L3) 31 1 -1 -0 29 -4 -6 R pars lumborum (L4) 32 1 1 -0 30 -7 6 L pars lumborum (L4) 32 1 -1 0 30 -7 -6 R il iocostalis lumborum 58 5 4 1 57 14 -1 L il iocostalis lumborum 58 5 -4 -1 57 14 1 R longissim us thoracis 93 7 4 0 91 23 -6 L longissimus thoracis 93 7 -4 -0 91 23 6 (continued) 97 Table 5.5 (continued) Force Moment (Nm) Compression Shear (N) --- N Flexion Lateral Twist N Anteroposterior f--- Lateral Muscle (continued) R quadratus lumborum 25 1 2 -0 25 -1 1 -- -- --L quadratus lumborum 25 1 -2 0 25 -1 -1 - R latissimus dorsi (L5) 15 1 1 -0 14 -1 -6 - L latissimus dorsi (L5) 15 1 -1 0 14 -1 6 R multifidus 1 28 1 1 1 26 6 9 -- - L multifidus 1 28 1 -1 -1 26 6 -9 -- -- R multifidus 2 28 1 1 0 28 6 0 -- L multifidus 2 28 1 -1 -0 28 6 0 - R psoas (L 1) 25 1 2 0 24 9 6 L psoas (L 1) 25 -1 -2 -0 24 9 -6 !-- - R psoas (L2) 25 -1 2 0 24 9 6 - --L psoas (L2) 25 -0 -2 -0 24 9 -6 - R psoas (L3) 25 -0 1 0 24 9 7 -- - L psoas (L3) 25 -0 -1 -0 24 9 -7 R psoas (L4) 25 -0 1 1 24 9 8 -- L psoas (L4) 25 -0 -1 -1 24 9 -8 -- Ligam ent , Anterior longitudinal 0 0 0 0 0 0 - Posterior longitudinal 86 2 0 0 261 44 -- - Ligam entum flavum 21 1 0 0 21 2 - R i ntertransverse 14 0 0 0 13 3 - - L i ntertransverse 14 0 -0 -0 13 3 - -I-- --R a rticular 74 2 1 1 65 40 - L --- a rticular 74 2 -1 -1 65 40 - '--- 98 Normal and Injury Mechanics of the Lumbar Spine 99 Table 5.5 (continued) Force Moment (Nm) Compression Shear (N) --- N Flexion Lateral Twist N Anteroposterior Lateral rLigam ent (continued) R articular 2 1 03 3 2 2 84 -3 - L articular 2 1 03 3 -2 -2 84 -3 - Interspinous 1 301 18 0 0 273 1 42 - --- Interspinous 2 345 14 0 0 233 268 - Interspinous 3 198 10 0 0 1 94 238 - - Supraspinous 592 41 0 0 591 79 - -- R lumbodorsal fascia 1 22 8 1 -0 1 09 -1 - L lumbodorsal fascia 1 22 8 -1 0 1 09 -1 - Passive Disc Gut, etc. - 9 0 0 - - - - 11 0 0 - - - -- by flexing at the hip, minimizing ligament and other passive tissue tension and relegating the moment generation responsibility to the musculature. An example in which the spine is flexed is presented later in this chapter. As described in chapter 4, the pars thoracis extensors are very effective l umbar spine extensors, given their large moment arms. Also, since the lifter's upper body is flexed, large reaction shear forces on the spine are produced (the rib cage is trying to shear forward on the pelvis). These shear forces are supported to a velY large degree by the pars lumborum extensor muscles. Furthermore, the abdominal m uscles are activated but produce a negligible contribution to moment/posture support. Why are they active? These muscles are activated to stabilize the spinal column, although this mild abdominal activity imposes a compression penalty to the spine. A more robust explanation of stabilizing mechanics is described in chapter 6. The preceding example demonstrates how diffusely the forces are distributed and illustrates how proper clinical interpretation requires anatomically detailed free body diagrams that represent reality (such as those incorporated into the virtual spine model). I believe that oversimplified free body diagrams have overlooked the important mechanical compressive and especially shear components of m uscular force. This has compromised the assessment of injUly mechanisms and the formulation of optimal therapeutic exercise. 1 00 Low Back Disorders Table 5.6 Musculature Components for Moment Generation of 450 Nm During Peak Loading for a Squat Lift of 27 kg (59.5 lb) Force Moment Compression Shear Muscle (N) (Nm) (N) (N) Rectus a bdominis 25 -2 24 5 External oblique 1 45 1 39 24 External oblique 2 43 -2 30 31 Internal oblique 1 14 1 14 -2 Internal oblique 2 23 -1 17 -1 6 Longissimus thoracis pars lumborum L4 862 35 744 -436 Longissimus thoracis pars lum borum L3 1 5 14 93 1 422 -5 18 Longissimus thoracis pars l umboru m L2 1 342 121 1 342 0 Longissimus thoracis pars lumborum L 1 1 302 1 1 0 1 302 0 Il iocostalis lumborum pars thoracis 369 31 369 0 Longissimus thoracis pars thoracis 295

25 295 0 Quadratus l um borum 393 16 386 74 Latissimus dorsi L5 1 1 2 6 79 -2 M ultifidus 1 1 36 8 1 34 18 M ultifidus 2 226 8 1 89 1 24 Psoas L 1 26 0 23 12 Psoas L2 28 0 27 8 Psoas L3 28 1 , 27 6 Psoas L4 28 1 27 5 Negative moments correspond to flexion, while negative shear corresponds to L4 shearing posteriorly on LS. Loads on the Low Back During Walking Thousands of low-level loading cycles are endured by the spine evelY day during walking. While the small loads in the low back during walking suggest it is a safe and tolerable activity, clinicians have fou nd that walking provides relief to some individuals but is painful to others. Recent work has suggested that walking speed affects spine mechanics and may account for these individual differences. During walking, the compressive loads on the lumbar spine of approximately 2.5 times body weight, 350 300 1: OJ 250 'iii 200 "0 .8 150 a 100 50 o 20 o 15 .10 :::; c: 0 > °fl « [) > :::; t c: 0 13 « 6 5 4 3 1 0 0 R-RA o R-EO o R-IO 10 20 30 40 50 60 70 80 Stride(%) 10 20 30 40 50 60 70 80 10 20 Stride(%) 30 40 50 Stride(%) 60 70 80 90 100 90 100 90 100 o :-- iii o R-LD o R-TES 10 20 30 40 50 Stride (%) 10 20 30 40 50 Stride(%) 60 70 60 70 o 10 20 30 40 50 60 70 R-LES Stride (%) 80 80 80 90 100 90 100 90 100 ES. ; I 0 10 20 30 40 50 60 70 80 90 100 R-MULT Stride(%) [) > :::; t c: .Q <0 ." 13 « 6 5 4 3 2 1 0 0 10 20 30 40 50 L-RA Stride(%) 60 70 ... - .. --;.--..------::.:: Fast -- - -- Normal ---- Slow 80 90 100 o L-EO 10 20 30 40 50 60 70 80 90 100 o L-IO o L-LD o L-TES 10 20 Stride(%) 30 40 50 Stride (%) 10 20 30 40 50 Stride (%) 10 20 30 40 50 Stride (%) 60 70 60 70 60 70 80 80 80 90 100 90 100 90 100 7 6 5 ", ,' ', :o 10 20 30 40 50 60 70 L-LES Stride (%) 80 90 100 o]] I I o L-MULT 10 20 30 40 50 60 70 80 90 100 Stride (%) Figure 5.7 Activation profiles (EMG signals) from 14 torso muscles during three speeds of walking with normal arm swing and normalized to RHC to RHC The muscle pairs are (RA) rectus abdominis, (EO) external oblique, (10) internal oblique, (LD) latissimus dorsi, (TES) thoracic erector spinae, (LES) lumbar erector spinae, and (MULT) multifidus. 1 03 1 04 Low Back Disorders Loads on the Low Back During Sitting - Nachemson (966), using intradiscal pressure measurements, documented the higher loads on the discs in various sitting postures compared to the standing posture. Normal sitting causes flexion in the lumbar spine, and people, if left alone, generally sit in a variety of flexed postures (Callaghan and McGill, 2001b). Sitting generally involves lower abdominal wall activity (particularly the deep abdomina ls) compared to standing and generally higher extensor activity with unsupported sitting (see Callaghan, Patia, and McGill, 1999 for walking and Callaghan and McGill, 2001b for sitting). Sitting slouched minimizes muscle activity, while sitting more upright requires higher activation of the psoas and the extensors (Juker, McGill, Kropf et al., 1998). Full flexion increases disc annulus stresses; this posture has produced disc herniations in the lab (e.g. , Wilder, Pope, and Frymoyer, 1 988). In fact, Kelsey (975) discovered a specific link between prolonged sitting and the incidence of herniation. More upright sitting postures, and the concomitant psoas and other muscle activation, impose additional compressive loads on the spine. Changing lumbar postures causes a migration of the loads from one tissue to another. Callaghan and McGill (2001b) suggested that no Single, ideal sitting posture exists; rather, a variable posture is recommended as a strategy to minimize the risk of tissue overload. Sitting Posture Despite the myth perpetuated in many ergonomic guidelines regarding a single "ideal" posture for sitting, the ideal sitting posture is one that continually changes, thus preventing any Single tissue from accumulating too much strain. Sitting strategies are presented in chapter 8. Loads on the Low Back During Flexion Exercises Very few studies (only our own that we are aware 01) have quantified the tissue loading on the low back tissues during various types of torso flexion exercises, although some have measured the EMG activity of selected muscles (e.g .. Flint, 1965; Halpem and Bleck, 1979; Jette, Sidney, and Cicutti, 1984). This type of

infolmation alone can proVide inSight into relative muscle challenge, but it is restrictive for guiding exercise prescription decisions because the resultant spine load is unknown. The goal is to challenge muscle at appropriate levels but in a way that spares the spine. Too many exercises are prescribed for back sufferers that exceed the tolerance of their compromised tissues. In fact, I believe that many commonly prescribed flexion exercises result, in so much spine compression that they will ensure that the person remains a patient. For example, the traditional sit-up imposes approximately 3300 N (about 730 lb) of compression on the spine (Axler and McGill, 1997). (Figure 5.8 illustrates psoas and abdominal muscle activation levels in a variety of flexion tasks, while figure 5.9 illustrates activation patterns with bent-knee situps, and figure 5.10 illustrates activation patterns with bent-knee curl-ups.) Note that muscle activation levels are expressed in normalized units (% MVC). This means that the activity is expressed as a percentage of what would be observed during a maximal voluntary contraction (MVC), thus quantifying activity in a physiological and functional context. Further, the spine is very flexed during the period of this load (McGill, 1998). The National Institute of Occupational Safety and Health (NIOSH) (981) has set the action limit for low back compression at 3300 N; repetitive loading above this level is linked with higher injury rates in workers, yet this is imposed on the spine with each repetition of the sit-up! Table 5.7 shows the quantification of a variety of flexion exercises. Cross curl-up Curl-up Isom side support Straight-leg sit-up Push-up from feet Bent-knee leg raise Oyn side support Bent knee sit-up Straight-leg raise Press-heel sit-up Isom r hand to I knee 0 Normal and Injury Mechanics of the Lumbar Spine 1 05 Many recommend performing sit-ups with the knees bent, the theory being that the psoas is realigned to reduce compressive loading, or perhaps the psoas is shortened on the length-tension relationship so that the resulting forces are reduced. After examining both of these ideas, we found them to be untenable. We placed a group of women who were small enough to fit into an MRI scanner and varied the knee and hip angles while they were supine (Santaguida and McGill, 1995). The psoas did not change its line of action, nor could it since it is attached to each vertebral body and transverse process (as the lumbar spine increases lordosis, the psoas follows this curve). The psoas does not change its role from a flexor to an extensor as a function of lordosis-this interpretation error occurred from models in which the psoas was represented as a straight line puller. In fact, the psoas follows the lordotic curve as the lumbar spine flexes and extends. Further, it is true that the psoas is shortened with hip flexion, but its activation level is higher during bent-knee sit-ups (Juker, McGill, Kropf et aI., 1998), not lower as has been previously thought. This is because the hip flexion torque must come from somewhere, and the shortened psoas must contract to higher levels of activation given its compromised length. Given that the sit-up imposes such a large compression load on the spine, regardless of the leg being bent or straight, the issue is not which type of sit-up should be recommended. Rather, situps should not be performed at all by most people. Far better ways exist to preserve the abdominal muscle challenge while imposing lower spine loads. Those who are training for health never need to perform a sit-up; those training 10 20 30 40 50 60 70 80 for performance may get better results by judio o • • o Percent MVC Psoas 1 Psoas 2 Rectus abd Ext oblique Int oblique Trans abd Ciously incorporating them into their routine. Figure S.B Activation of the psoas and the abdominal muscles in a variety of flexion tasks from a group of highly trained subjects (five men and three women). While part III of this book (Low Back Rehabilitation) offers specific preferred exercises and challenges to specific muscles, a few flexion exercises will be reviewed here. First, hanging with the arms from an overhead bar and flexing the hips to raise the legs is often thought to impose low spine loads because the body is hanging in tension-not compression. This is faulty logiC.

This hanging exercise generates well over 100 Nm of abdominal torque (Axler and McGill, 1997). This produces almost maximal abdominal activation, which in turn imposes compressive forces on the spine (see table 5.7). Similar activation levels can be achieved with the side bridge (shown later and discussed in detail) with lower spine loads. 106 Low Back Disorders 1 00 80 60 40 o > 20 o 2 3 4 -- Psoas 1 ind --Rectus abd sur - - - Ext oblique i nd ..... I nt oblique ind --Trans abd ind 'I I 5 6 1\ , ", \ I v I, ""\ 7 Time (s) 8 9 (" ,I \ t'\ J '- J \ - I' "'I \' "" \ , 10 11 12 , , 13 14 Figure 5.9 Activation-time histories of the same subjects as in figure 5.8 performing a bent-knee sit-up. Surface and indwelling electrodes are indicated. Having stated this, those not interested in sparing their back and who are training with performance objectives may benefit from the high psoas challenge, together with rectus abdominis and oblique activity. Clearly, the curl-up primarily targets the rectus (both upper and lower), and generally other exercises should be performed to train the obliques. Some have suggested a twisting curl-up to engage the obliques, but this results in a poor ratio of oblique muscle challenge to spine compression compared to the side bridge exercise (Axler and McGill, 1997)-making the side bridge a preferred exercise. Loads on the Low Back During Extension Exercises As with the flexion exercises discussed in the previous paragraph, plenty of EMGbased studies have explored extension exercises, but only one attempted to quantify the resulting tissue loads. Exercise prescriptions will not be successful if the spine loading is not constrained for bad backs. Using the virtual spine approach, Callaghan, u Normal and Injury Mechanics of the Lumbar Spine 1 07 1 0 80 0 ----------. 60 ,K t t 40 20 o .\ . . "" I . . _ - ,,- -,,-,,' _ :::.' '. ,, . . . -:'. . ,' !" -':':-"" ' " " _ .• _ _ _ / ::./ . .I _ .. :'p _ . . . 2 Psoas 1 ind Rectus abd sur Ext oblique ind I nt oblique ind Trans abd ind 3 4 5 6 Time (s) 7 8 9 10 11 Figure 5.10 Activation-time histories for the same group performing curl-ups. Gunning, and McGill (1998) attempted to rank extension exercises on the muscle challenge, the resultant spine load, and their optimal ratio. The key to preserving a therapeutic muscle activation level while minimizing the spine load is to activate only one side of the spine musculature at a time. The previous muscle anatomy section in chapter 4 describes the functional separation of the thoracic and lumbar portions of the longissimus and iliocostalis. For the purposes of this discussion, we can think of the extensors in four sections-right and left thoracic portions and right and left lumbar portions. The common extension task of performing torso extension with the legs braced and the cantilevered upper body extending over the end of a bench or Roman chair (figure 5.1 1a) activates all four extensor groups and typically imposes over 4000 N (about 890 lb) of compression on the spine. Even worse is the commonly prescribed back extension task in clinics, in which the patient lies prone and extends the legs and outstretched arms; this again activates all four extensor sections but imposes up to 6000 N (over 1 300 lb) on a hyperextended spine (figure 5.1 1b). This is not justifiable for any patient! Several variations of exercise technique can preserve activation 1 08 Low Back Disorders Table 5.7 Low Back Moment, Abdominal Muscle Activity, and Lumbar Compressive Load During Several Types of Abdominal Exercises Muscle activation Moment Rectus abdominis External Compression (Nm) (% MVC) oblique (N) Straight-leg sit-up 1 48 121 70 3 506 B ent-leg sit-up 1 54 1 03 70 3350 Curl-up, feet anchored 92 87 45 2009 Curl-up, feet free 81 67 38 1 991 Qua rter sit-up 1 1 4 78 42 2392 Straig ht-leg raise 1 02 57 35 2525 Bent-leg raise 82 35 24 1 767 Cross-kn ee curl-up 112 89 67 2964 Hanging, straight l eg 1 07 1 1 2 90 2805 Hanging, b ent l eg 84 78 64 3 3 13 Isometric side bridge 72 48 50 2585 MVC contractions were isometric. Activation values higher than 100% are often seen during dynamic exercise. in portions of the extensors and greatly spare the spine of high load. For example, kneeling on all fours and

extending one leg at the hip generally activates one side of the lumbar extensors to over 20% of maximum and imposes only 2000 N of compression (figure 5.l lc). Performing the birddog, in which the opposite arm is extended at the shoulder while the leg is raised (figure 5.1 1d), adds activity to one side of the thoracic extensors (generally around 30-40% of maximum) and contains the spine load to about 3000 N. In addition, the special techniques shown for this exercise in the exercise prescription chapter (chapter 13) attempt to enhance the motor control system to groove stabilizing patterns. For data describing these exercises, see table 5.B. Dubious Lifting Mechanisms In the 1950s and 1 960s spine biomechanists faced a paradox. The simple spine models of the day predicted that the spine would be crushed to the point of injury during certain lifting tasks, yet when people performed those tasks, they walked away uninjured. This motivated several research groups to theorize about mechanisms that could unload compressive stresses from the spine. Researchers proposed three major mechanisms: the intra-abdominal pressure mechanism, the lumbodorsal facia mechanism, and the hydraulic amplifier. Although none has survived scrutiny, clinical vestiges still remain. Nonetheless, some components provided insight for subsequent study and led to the understanding that we have today. For this reason they will be reviewed briefly here. .... a c d Figure 5.1 1 Specific extension exercises quantified for muscle activation and the resultant spine load (shown in table 5.8): (a) trunk extension, (b) prone leg and trunk extension, (c) single-leg extension, and (d) single-leg and contralateral arm extension (birddog). Table 5.8 Mean Activation Levels (± 1 SO) of 14 EMG Channels for 13 Subjects Performing a Variety of Extensor-Dominant Exercises (Expressed As a Percentage of MVC) Extension Right leg Left leg Electromyographic and and Trunk Calibration channela Right leg Left leg left arm right arm and legs Trunk posture Rig ht RA X 3.3 2.7 4.0 3.5 4.7 3.1 1 .4 SO 2.4 1 .9 2.0 2.0 2.2 1 .8 1 .0 Right EO X 8.4 4.9 1 6.2 5.2 4.3 3.7 1 .0 SO 4.9 1 .5 6.0 2.3 2.5 1 .7 0.6 Right 10 X 1 2.0 8.2 1 5.6 1 2.0 1 2.1 1 2.7 1 .9 SO 6.8 2.5 8.2 4.2 1 0.1 1 0.8 1 .2 Right LO X 8.1 5.8 1 2.0 1 2.5 1 1 .2 6.5 5.9 SO 5 .4 3.5 9.6 6.2 4.3 4.0 8.5 Rig ht TES X 5.7 1 3.7 1 1 .5 46.8 66.1 45.4 2 1 .0 SO 2.0 7.5 6.6 29.3 1 8.8 1 0.6 9.0 (con tinued) ' 09 Table 5.8 (continued) - Extension Right leg Left leg Electromyographic and and Trunk Calibration channel" Right leg Left leg left arm right arm and legs Trunk posture Right LES X 1 9.7 11.7 28.4 1 9.4 59.2 5 7.8 2 1 .3 - SO -- 9.1 4.9 1 0.2 11.0 1 1 .7 8.5 4.6 - - Right MF X 21.9 1 0.8 31.5 1 6.1 51.9 47.5 1 6.4 SO 6.3 6.0 8.2 1 2.0 1 4.7 1 2.3 5.6 - - - - Left RA X 4.3 3 .6 4.4 4.2 6.5 3.7 2.2 SO 3.4 3 .6 3.8 3.9 3.4 2.4 2.1 - - Left EO X 5.4 9.0 6.2 1 5.9 6.3 5.2 1 .8 SO 2.0 3 .8 2.5 6.6 3.2 - 5.2 1 .0 Left 10 X 1 6.0 11.3 22.6 1 5.2 11.0 1 2.5 1 .6 SO 8.6 7.0 9.2 6.7 5.9 6.1 1 .3 1 - Left LO X 4.5 5 .0 1 0.7 6.2 9.2 5.1 6.1 SO 4.3 4.5 1 8.2 4.4 5.1 4.1 8.5 -- - Left TES X 1 5.0 4.5 42.9 1 0.5 63.6 41 .6 2 1 .2 SO - - 7.5 2.0 20.5 5 .9 22.7 1 0.0 9.8 r - - Left LES X 1 1 .3 1 6.8 1 9.5 ' 25.5 56.8 57.0 23.3 SO 6.6 4.5 7.4 7.3 1 4.5 1 4.7 - 8.4 Left MF X 11.9 I 22.3 1 6.6 33.8 5 7.3 53.3 1 8.7 SO 7.0 6.1 7.2 6.7 1 1 .4 1 2.0 I 4.3 a Electromyographic channel: RA = rectus abdominis muscle, EO = external oblique muscle, 10 = internal oblique muscle, LD = latissimus dorsi muscle, TES = thoracic erector spinae muscle, LES = lumbar erector spinae muscle, MF = multifidus muscle. Calibration posture: standing, trunk flexed 60°, neutral lumbar posture, 10 kg (22 Ib) held in hands with arms hanging straight down. 1 1 0 Intra-Abdominal Pressure Normal and Injury Mechanics of the Lumbar Spine 111 Does intra-abdominal pressure (IAP) play an important role in the support of the lumbar spine, especially during strenuous lifting, as has been claimed for many years? Anatomical accuracy in representation of the involved tissues has been influential in this debate. Further, research on lifting mechanics has formed a cornerstone for the prescription of abdominal belts for industrial workers and has motivated the prescription of abdominal strengthening programs. Many researchers have advocated the use of intra-abdominal

pressure as q. mechanism to directly reduce lumbar spine compression (Beam, 1961; Thomson, 1988). However, some researchers believe that the role of JAP in reducing spinal loads has been overemphasized (Grew, 1980; Krag et aI., 1986). Anatomical Consistency in Examining the Role of Intra-Abdominal Pressure Morris, Lucas, and Bresler (961) first operationalized the mechanics of the original proposal into a model and described it as follows. Pressurizing the abdomen by closing the glottiS and bearing down during an exeltion exerts hydraulic force down on the pelviC floor and up on the diaphragm, creating a tensile effect over the lumbar spine or at least alleviating some of the compression. Missing in the early calculations of this hydraulic potential was the full acknowledgment of the necessalY abdominal activity (contacting the abdominal wall imposes extra compression on the spine). But evaluation of the trade-off between the extra abdominal muscle compression and the hydraulic relief depended on the geometrical assumptions made. Some of these assumptions appear to be outside of biological reality. In fact, experimental evidence suggests that somehow, in the process of building up lAP, the net compressive load on the spine is increased! Krag and coworkers (986) observed increased low back EMG activity with increased lAP during voluntalY Valsalva maneuvers. Nachemson and Morris (964) and Nachemson, Andersson, and Schultz (986) showed an increase in intradiscal pressure during a Valsalva maneuver, indicating a net increase in spine compression with an increase in lAP, presumably a result of abdominal wall musculature activity. In our own investigation, in which we used our virtual spine model, we noted that net spine compression was increased from the necessary concomitant abdominal activity to increase lAP. Furthermore, the size of the cross-sectional area of the diaphragm and the moment arm used to estimate force and moment at the lower lumbar levels, produced by lAP, have a major effect on conclusions reached about the role of lAP (McGill and Norman, 1987). The diaphragm surface area was taken as 243 cm2 , and the centroid of this area was placed 3.8 cm anterior to the center of the T12 disc (compare these values with those used in other studies: 5 1 1 cm2 for the pelvic floor, 465 cm2 for the diaphragm, and moment arm distances of up to 11.4 cm, which is outside the chest in most people). During squat lifts, the net effect of the involvement of the abdominal musculature and lAP seems to be to increase compression rather than alleviate joint load. (A detailed description and analysis of the forces are in McGill and Norman, 1 987). This theoretical finding agrees with the experimental evidence of Krag and colleagues (986), who used EMG to evaluate the effect of reducing the need for the extensors to contract (they didn't), and of Nachemson and colleagues (986), who documented increased intradiscal pressure with an increase in lAP. Role Of lAP During Lifting The generation of appreciable lAP during load-handling tasks is well documented. The role of lAP is not. Farfan (973) suggested that lAP creates a pressurized visceral cavity to maintain the hooplike geometry of the abdomina Is. In recent work in which they measured the distance of the abdominals to the spine (moment arms), McGill, 1 12 Low Back Disorders Juker, and Axler (996) were unable to confirm substantial changes in abdominal geometlY when activated in a standing posture. However, the compression penalty of abdominal activity cannot be discounted. The spine appears to be well suited to sustain increased compression loads if intrinsic stability is increased. An unstable spine buckles under extremely low compressive loads (e.g. , approximately 20 N, or about 5 Ib) (Lucas and Bresler, 1 961). The geometry of the spinal musculature suggests that individual components exert lateral and anterior/posterior forces on the spine that can be thought of as guy wires on a mast to prevent bending and compressive buckling (Cholewicki and McGill, 1996). As well, activated abdomina Is create a rigid cylinder of the trunk, resulting in a stiffer structure. Recently, both Cholewicki and colleagues (999) and Grenier and colleagues (in

press) documented increased torso stiffness during elevated lAP even when accounting for similar abdominal wall contraction levels. Thus, although the increased lAP commonly observed during lifting and in people experiencing back pain does not have a direct role in reducing spinal compression or in adding to the extensor moment, it does stiffen the trunk and prevent tissue strain or failure from buckling. Lumbodorsal Fascia Recent studies have attributed various mechanical roles to the lumbodorsal fascia (LDF). In particular, some have suggested that the LDF reduces spine loads-solving the paradox noted earlier. In fact, some have recommended lifting postures based on various interpretations of the mechanics of the LDF. Gracovetsky, Farfan, and Lamy (981) originally suggested that lateral forces generated by internal oblique and transverse abdominis are transmitted to the LDF via their attachments to the lateral border. They also claimed that the fascia could support substantial extensor moments. FUl1her, lateral tension from abdominal wall attachments was hypothesized to increase longitudinal tension from Poisson's effect, causing the posterior spinous processes to move together resu lting in lumbar extension. This sequence of events formed an attractive proposition because the LDF has the largest moment arm of all extensor tissues. As a result, any extensor forces within the LDF would impose the smallest compressive penalty to vertebral components of the spine. Three independent studies, however, examined the mechanical role of the LDF and collectively questioned the idea that the LDF could support substantial extensor moments (Macintosh and Bogduk, 1 987; McGill and Norman, 1988; Tesh, Dunn, and Evans, 1987). As previously noted, regardless of the choice of LDF activation strategy, the LDF contribution to the restorative extension moment was negligihle compared with the much larger low back reaction moment required to support a load in the hands. Its function may be that of an extensor muscle retinaculum (Bogduk and Macintosh, 1984). Hukins, Aspden, and Hickey (990) proposed on theoretical grounds that the LDF acts to increase the force per unit of cross-sectional area that muscle can produce by up to 30%. They suggested that this increase in force is achieved by constraining bulging of the muscles ⬜hen they shorten. This contention remains to he proven. Tesh, Dunn, and Evans (987) suggested that the LDF may be important for supporting lateral bending. Furthermore, there is no question that the LDF is involved in enhancing stahility of the lumbar column (see chapter 6). No doubt, complete assessment of these notions will be pursued in the future. HydraUlic Amplifier The final mechanism hypothesized to unload compressive stresses from the spine was the hydraulic amplifier. This hybrid mechanism depends on three notions. First, the elevated lAP preserves the hooplike geometlY of the abdominal wall during exertion. Normal and Injury Mechanics of the Lumbar Spine 113 The lAP must also exert hydraulic pressure posteriorly over the spine and presumably through to the underside of the lumbodorsal fascia. Finally, as the extensor muscle mass contacts, it was proposed to "bulk" upon shortening, again increasing the hydraulic pressure under the fascia. The biomechanical attraction of the pressure under the fascia is that any longitudinal forces generated in the fascia reduce the need for the underlying muscles to contribute extensor forces, thereby lowering the compressive load on the spine. Both of these proposals were dismissed. Given the size of the fascia, hydraulic pressures would have to reach levels of hundreds of mmHg. Pressures of this magnitude simply are not observed during recording (Carr et al., 1985). Moreover, the presence or absence of lAP makes little difference on the hooplike geometry of the abdominal wall (McGill, Juker, and Axler, 1 996), as this is more modulated by the posture. lAP, LDF, and Hydraulic Amplifier: A Summary lAP, the mechanical role of the LDF, and the existence of the hydraulic amplifier were proposed to account for the paradox that people were able to perform lifts that the simple models suggested would crush their spines. Yet, although both lAP and the LDF appear to play

some role in lifting, none of the three proposed mechanisms was a tenable explanation for the paradox of uncrushed spines under heavy loading, whether considered separately or combined with the other two. The problem lay in the simple models of three and four decades ago. Not only were the rather complex mechanics not represented with the necessalY detail, but also the strength of the tissues to bear load was also quite underestimated in the early tests that used old cadaveric samples that were crushed in artificially stiff rams of materials-testing machines that caused failure too early. Other Important Mechanisms of Normal Spine Mechanics Several other features of spine mechanics influence function and ultimately underpin strategies for injury prevention and rehabilitation. The most important are presented here. Biomechanics of Diurnal Spine Changes Most people have experienced the ease of taking off their socks at night compared to putting them on in the morning. The diurnal variation in spine length (the spine being longer after a night's bed rest), together with the ability to flex forward, has been well documented. Reilly, Tynell, and Troup (984) measured losses in sitting height over a day of up to 19 mm. They also noted that approximately 54% of this loss occurred in the first 30 minutes after rising. Over the course of a day, hydrostatic pressures cause a net outflow of fluid from the disc, resulting in narrowing of the space between the vertebrae, which in turn reduces tension in the ligaments. When a person lies down at night, osmotic pressures in the disc nucleus exceed the hydrostatic pressure, causing the disc to expand. Adams, Dolan, and Hutton (987) noted that the range of lumbar flexion increased by 5-60 throughout the day. The increased fluid content after rising from bed caused the lumbar spine to be more resistant to bending, while the musculature did not appear to compensate by restricting the bending range. Adams and colleagues estimated that disc-bending stresses were increased by 300% and ligament stresses by 80% in the morning compared to the evening; they concluded that there is an increased risk of injury to these tissues when bending fOlward early in the morning. Recently, Snook and colleagues (998) demonstrated that simply avoiding full lumbar flexion in the morning reduced back symptoms. We are beginning to understand the mechanism. 1 14 Low Back Disorders Exercise People should not undertake spine exercises-particularly those that require full spine flexion or bending-just after rising from bed, given the elevated tissue stresses that result. This would hold true for any occupational task requiring full spine range of motion. Spinal Memory The function of the spine is modulated by certain previous activity. This occurs because the loading histOlY determines disc hydration (and therefore the size of the disc space and disc geometlY)' which in turn modulates ligament rest length, joint mobility, stiffness, and load distribution. Consider the following scenario: McKenzie (1979) proposed that the nucleus within the annulus migrates anteriorly during spinal extension and posteriorly during flexion. McKenzie's program of passive extension of the lumbar spine (which is currently popular in physical therapy) was based on the supposition that an anterior movement of the nucleus would decrease pressure on the posterior portions of the annulus, which is the most problematic site of herniation. Because of the viscous properties of the nuclear material, such repositioning of the nucleus is not i mmediate after a postural change but rather takes time. Krag and coworkers (1987) observed anterior movement of the nucleus during lumbar extension, albeit q uite minute, from an elaborate experiment that placed radio-opaque markers in the nucleus of cadaveric lumbar motion segments. Whether this observation was caused simply by a redistribution of the centroid of the wedge-shaped nuclear cavity moving forward with flexion or was a movement of the whole nucleus remains to be seen. Nonetheless, hydraulic theolY would suggest lower bulging forces on the posterior annulus if the nuclear centroid moved anteriorly d uring extension. If compressive forces were applied

to a disc in which the nuclear material were still posterior (as in lifting immediately after a prolonged period of flexion), a concentration of stress would occur on the posterior annulus. While this specific area of research needs more development, a time constant seems to be associated with the redistribution of nuclear material. If this result is correct, it would be unwise to lift an object immediately following prolonged flexion, such as sitting or stooping (e.g. , a stooped gardener should not stand erect and immediately lift a heavy object). Furthermore, Adams and Hutton (1988) suggested that prolonged full flexion may cause the posterior ligaments to creep, which may allow damaging flexion postures to go unchecked if lordosis is not controlled during subsequent lifts. In a study of posterior passive tissue creep while sitting in a slouched posture, McGill and Brown (1992) showed that over the 2 minutes following 20 minutes of full flexion, subjects regained only half of their intervertebral joint stiffness. Even after 30 minutes of rest, some residual joint laxity remained. This finding is of particular importance for individuals whose work is characterized by cyclic bouts of full end range of motion postures followed by exertion. Before lifting exertions following a stooped posture or after prolonged sitting, a case could be made for standing or even consciously extending the spine for a short period. Allowing the nuclear material to "equilibrate," or move anteriorly to a position associated with normal lordosis, may decrease forces on the posterior nucleus in a subsequent lifting task. Ligaments will regain some protective stiffness during a short period of lumbar extension. In conclusion, rhe anatomy and geometry of the spine are not static. Much research remains to be done to understand the importance of tissue loading history on subsequent biomechanics, rehabilitation therapies, and injury mechanics. Functional SlnnItll" Normal and Injury Mechanics of the Lumbar Spine 115 of Spinal Mem It would appear protective to avoid loading immediately after a bout of prolonged flexion. In the occupational world this has relevance to ambulance drivers, for example, who drive to an accident scene without the luxury of time to warm up (or reset the passive tissues) before , lifting. They would be wise to sit with a lumbar pad to avoid lumbar flexion and the associated creep. The athletic world provides good examples as well, such as Sitting on the bench before engaging in play. Those with sensitive backs would do well to avoid siting on the bench with a flexed lumbar spine while waiting to perform. We recently quantified the loss of compliance in the lumbar spine with bench sitting between bouts of athletic performance (Green, Grenier, and McGill, in press) in elite volleyball players. Viscosity is also a consideration in prolonged postures since "internal friction" increases with prolonged static postures. Sitting in this way, and the associated changes in stiffness and viscosity, is detrimental to athletes' performance and increases their risk of injury. We will address this issue more completely in chapters 8 and 9. Anatomical Flexible Beam and Truss: Muscle Cocontraction and Spine Stability The osteoligamentous spine is somewhat of an anatomical paradox: it is a weightbearing, upright, flexible rod. Observationally, the ability of the joints of the lumbar spine to bend in any direction is accomplished with large amounts of muscle coactivation. Such coactivation patterns are counterproductive to generating the torque necessary to support the applied load. Coactivation is also counterproductive to minimize the load penalty imposed on the spine from muscle contraction. Researchers have postulated several ideas to explain muscular coactivation: the abdominals are involved in the generation of intra-abdominal pressure (Davis, 1959) or in providing support forces to the lumbar spine via the lumbodorsal fascia (Gracovetsky, Farfan, and Lamy, 1 981). These ideas have not been without opposition (see previous sections). Another explanation for muscular coactivation is tenable. A ligamentous spine will fail under compressive loading in a buckling mode, at about 20 N (about 5 Ib) (Lucas and Bresler, 1961). In other words, a bare spine is unable to bear compressive load!

The spine can be likened to a flexible rod that will buckle under compressive loading. However, if the rod has guy wires connected to it, like the rigging on a ship's mast, although more compression is ultimately experienced by the rod, it is able to bear much more compressive load since it is stiffened and therefore more resistant to buckling. The cocontracting musculature of the lumbar spine (the flexible beam) can perform the role of stabilizing guy wires (the truss) to each lumbar vertebra, bracing it against buckling. Recent work by Crisco and Panjabi (990), together with Cholewicki and McGill (996), Cholewicki, Juluru, and McGill (999), and Gardner-Morse, Stokes, and Laible (995) has begun to quantify the influence of muscle architecture and the necessary coactivation on lumbar spine stability. The architecture of many torso muscles is especially suited for the role of stabilization (Macintosh and Bogduk, 1987; McGill and Norman, 1987) . • :0 ___u_c_u_a_r__o_c_o_n_tr_a_c_t_o_n_ In order to invoke this antibuckling and stabilizing mechanism when lifting, one could j ustify lightly cocontracting the musculature to minimize the potential of spine buckling. 1 16 Low Back Disorders Injury Mechanisms Many clinicians, engineers, and ergonomists believe that reducing the risk of low back injury involves the reduction of applied loads to the various anatomical components at risk of injury. Without question, reduction of excessive loads is beneficial, but this is an overly simplistic view. Optimal tissue health requires an envelope of loading, not too much or too little. While some occupations require lower loads to reduce the risk, in sedentary occupations, the risk can be better reduced with more loading, and valying the nature of the loading. To decide which is best, the clinician must have a thorough understanding of the biomechanics of injury, which comes in two parts. First is a brief review of the injury mechanisms of individual tissues; second is a description of the injury pathogenesis and several injury scenarios. Also needed is an understanding of generic situations for low back tissue damage described in the tissue injury primer at the end of chapter 1. Summary of Specific Tissue Injury Mechanisms This section provides a very brief description of damage from excessive load. All injuries noted are known to be accelerated with repetitive loading. • End plates. Schmorl's nodes are thought to be healed end-plate fractures (VernonRoberts and Pirie, 1 973) and pits that form from localized underlying trabecular bone collapse (Gunning, Callaghan, and McGill, 2001) and which are linked to trauma (Aggrawall et al., 1979). In fact, Kornberg (1988) documented (via MRI) traumatic Schmorl's node formation in a patient following forced lumbar flexion that resulted in an injury. People apparently are not born with Schmorl's nodes; their presence is associated with a more active lifestyle (Hardcastle, Annear, and Foster, 1992). Under excessive compressive loading of spinal units in the laboratory, the end plate appears to be the first structure to be injured (Brinckmann, Biggemann, and Hilweg, 1 988; Callaghan and McGill, 200la). Studies have revealed end-plate avulsion under excessive anterior/posterior shear loading. • Vertebrae. Vertebral cancellous bone is damaged under compressive loading (Fyhrie and Schaffler, 1994) and often accompanies disc herniation and annular delamination (Gunning, Callaghan, and McGill, 2001). • Disc annulus. Several types of damage to the disc annulus appear to occur. Classic disc herniation appears to be associated with repeated flexion motion with only moderate compressive loading required (Callaghan and McGill, 2001a) and with full flexion with lateral bending and twisting (Adams and Hutton, 1985; Gordon et al., 1 99 1). Yingling and McGill (1999a and 1999b) documented avulsion of the lateral annulus under anterior/posterior shear loading. • Disc nucleus. While Buckwalter (1995), when referring to the disc nucleus, stated that "no other musculoskeletal soft tissue structure undergoes more dramatic alterations with age," the relationships among loading, disc nutrition, decreasing concentration of viable cells, accumulation of degraded matrix molecules, and fatigue failure

of the matrix remain obscure. However, recently Lotz and Chin (2000) documented that cell death (apoptosis) within the nucleus increases under excessive compressive load. Interestingly, these changes are generally not detectable or diagnosable in vivo. • Neural arch (posterior bony elements). Spondylitic fractures are thought to occur from repeated stress-strain reversals associated with cyclic full flexion and extension (Burnett et al., 1 996; Hardcastle, Annear, and Foster, 1992). Cripton and colleagues (1995) and Yingling and McGill (1999a) also documented that excessive shear forces can fracture parts of the arch. Normal and Injury Mechanics of the Lumbar Spine 117 • Ligaments. Ligaments seem to avulse at lower load rates but tear in their midsubstance at higher load rates (Noyes, DeLucas, and Torvik, 1994). McGill (997) hypothesized that landing on the buttocks from a fall will rupture the interspinous complex given the documented forces (McGill and Callaghan, 1999) and joint tolerance. Falling on the behind increases the risk for prolonged disability (Troup, Martin, and Lloyd, 1981), which is consistent with the prolonged length of time it takes for ligamentous tissue to regain structural integrity, when compared with other tissues (Woo, Gomez, and Akeson, 1 985). Reducing Tissue Damage Summarizing injury pathways from in-vitro testing, evidence suggests that reduction in specific tissue damage could be accomplished by doing the following: 1. Reducing peak (and cumulative) spine compressive loads to reduce the risk of end-plate fracture 2. Reducing repeated spine motion to full flexion to reduce the risk of disc herniation (reducing spine flexion in the morning reduces symptoms) 3. Reducing repeated full-range flexion to full-range extension to reduce the risk of pars (or neural arch) fracture 4. Reducing peak and cumulative shear forces to reduce the risk of facet and neural arch damage 5. Reducing slips and falls to reduce the risk of passive collagenous tissues such as ligaments 6. Reducing the length of time sitting, particularly exposure to seated vibration, to reduce the risk of disc herniation or accelerated degeneration Injury Mechanics Involving the Lumbar Mechanism Many researchers have established that too great a load placed on a tissue will result in injllly. Epidemiological studies (Hilkka et a!., 1990; Marras et a! ., 1993; Norman et a!., 1998; Videman, Nurminen, and Troup, 1990) have proven this notion by identifying peak loading measures (i.e., shear, compression, trunk velocity, extensor moment, heavy work, etc.) as factors that explain the frequency and distribution of reporting of back pain or increased risk of back injury. However, the search for direct evidence that links spine load with occupational LBDs may have been hampered by focusing on too narrow a range of variables. Researchers have paid a massive amount of attention, for example, to a single variable-namely, acute, or single maximum exposure to, lumbar compression. A few studies have suggested that higher levels of compression exposure increased the risk of LBD (e.g., Herrin et a! ., 1986), although the correlation was low. Yet some studies show that higher rates of LBDs occur when levels of lumbar compression are reasonably low. What other mechanical variables modulate the risk of LBDs? As noted in chapter 1, they are as follows: • Too many repetitions of force and motion and/or prolonged postures and loads have also been indicated as potential injuly- or pain-causing mechanisms. • Cumulative loading (i.e., compression, shear, or extensor moment) has been identified as a factor in the reporting of back pain (Kumar, 1990; Norman, Wells, and Neumann, 1998). • Cumulative exposure to unchanging work has been linked to reporting of low back pain (Holmes, H ukins, and Freemont, 1993) and intervertebral disc injury (Videman, Nurminen, and Troup, 1990). 1 18 Low Back Disorders Many personal factors appear to affect spine tissue tolerance, for example, age and gender. In a compilation of the available literature on the tolerance of lumbar motion units to bear compressive load, Jager and colleagues (1991) noted that when males and females are matched for age, females are able to sustain only

approximately two thirds of the compressive loads of males. Furthermore, Jager and colleagues' data showed that, within a given gender, a 60-year-old was able to tolerate only about two thirds of the load tolerated by a 20-year-old. To complicate the picture, Holm and Nachemson (1983) showed that increased levels of motion are beneficial in providing nutrition to the structures of the intervertebral disc, while much of our lab's research has demonstrated that too many motion cycles (to full flexion) resulted in intervertebral disc herniation. Buckwalter (1995) associated intervertebral disc degeneration with decreased nutrition. Meanwhile, Videman and colleagues (1990) showed that too little motion from sedentary work resulted in intervertebral disc injury. While workers who performed heavy work were also at increased risk of developing a spine injury, workers who were involved in varied types of work (mixed work) had the lowest risk of developing a spine injury (Videman, Nunninen, and Troup, 1990). This presents the idea that too little motion or load, or too much motion or load can modulate the risk of spinal injLlly. In contrast, Hadler (1991) claimed that the incidence of back injury has not declined over the past 25 years, even after increased research and resources dedicated to the area over that time frame. Hadler suggested that the focus be turned from biomechanical causes of injLllY to developing more "comfortable" workplaces. However, the work described thus far in this text has clearly documented links to mechanical variables. Clearly, LBD causality is often extremely complex with all sorts of factors interacting. We will consider some of those factors in the following sections. nW'IPl'lnp Into Practice While the notion that too much of any activity (whether it be sitting at a desk or loading heavy boxes onto pallets) can be harmful is widely accepted, it is rarely taken into account in practice. For example, industrial ergonomics has not yet wholeheartedly embraced the idea that not all jobs need to be made less demanding, that some jobs need much more variety in the patterns of musculoskeletal loading, or that there is no such thing as "best posture" for sitting. It is time for the profession as a whole to remember that in any job the order and type of loading should be considered and the demand on tissues should be varied. All sedentary workers should be taught, for example, to adopt a variable posture that causes a migration of load from tissue to tissue, reducing the risk of troubles. Stoop Versus Squat in Lifting Injury Risk While the scientific method can prove that a phenomenon is possible if observed, failure to observe the expected result does not eliminate the possibility. One may only conclude that the experiment was insensitive to the particular phenomenon. The following discussion, as with many in this book, attempts to incorporate this limitation and temper it with clinical wisdom. In a previous section, lifting with the torso flexing about the hips rather than flexing the spine was analyzed and described. Specifically, the lifter elected to maintain a neutral lumbar posture rather than allowing the lumbar region to flex. Here we will reexamine the lifting exercise, but the lifter will flex the spine sufficiently to cause the posterior ligaments to strain. This lifting strategy (spine flexion) has quite dramatic effects on shear loading of the intervertebral column and the resultant injury risk. The Normal and Injury Mechanics of the Lumbar Spine 119 dominant direction of the pars lumborum fibers of the longissimus thoracis and iliocostalis lumborum muscles when the lumbar spine remained in neutral lordosis (page 64) caused these muscles to produce a posterior shear force on the superior vertebra. In contrast, with spine flexion the strained interspinous ligament complex generates forces with the opposite obliquity and therefore imposes an anterior shear force on the superior vettebra (see figures 5.12 and 5.13, a-d). Figure 5.1 2 This gardener appears to be adopting a fully flexed lumbar spine. Is this wise posture? The force analysis in figure 5.1 3 suggests it is not. Let's examine the specific forces that result from flexing the lumbar spine. The recruited ligaments appear to contribute to the anterior shear force so that shear force levels are likely to exceed 1 000 N (

224 lb). Such large shear forces are of great concern from an injury risk viewpoint. However, when a more neutral lordotic posture is adopted, the extensor musculature is responsible for creating the extensor moment and at the same time provides a posterior shear force that SUppOltS the anterior shearing action of gravity on the upper body and handheld load. The a joint shear forces are reduced to about 200 N (about 45 lb). Thus, using muscle to support the moment in a more neutral posture rather than being fully flexed with ligaments supporting the moment greatly reduces shear loading (see table 5.9). Quantification of the risk of injUly requires a comparison of the applied load to the tolerance of the tissue. Cripton and colleagues (1995) found the shear tolerance of the spine to be in the neighborhood of 2000-2800 N in adult cadavers, for onetime loading. Recent work by Yingling and McGill (1999a and 1 999b) on pig spines has shown that load rate is not a major modulator of shear tolerance unless the load is very ballistic, such as what might occur during a slip and fall. This example b Figure 5.1 3 These original computer image bitma;Js from the experiment conducted around 1 987 illustrate the fully flexed spine (aJ that is associated with myoelectric silence in the back extensors and strained posterior passive tissues and high shearing forces on the lumbar spine (from both reaction shear on the upper body and interspinous ligament strain (see figure 4.25). A more neutral spine (bJ posture recruits the pars lumborum muscle groups ... (continued) z CI> 2 0 LL c z CI> 2 d 700 600 500 400 300 200 1 00 0 0 4 3 2 Muscle forces Flexion/extension Rect ab -+- Ext oblique -+- I nt oblique -0- Lumbar erectors Lat dorsi ---<>- Thoracic erectors .. 2 3 4 Compression and Shear Force .. (f ..   ,.---------- I. "I CMP -+- SHRS 5 o o 2 3 4 5 Figure 5.1 3 (continued) ... (c) to support the reaction shear (see figure 4.1 9) and thus reduces total joint shear (d) (to approximately 200 N in this example). Reprinted from Journal of Biomechanics, 30(5), S.M. McGill,"Invited paper: Biomechanics of low back injury: Implications on current practice and the clinic," 465-475, 1997, with permission from Elsevier Science .. 1 20 Table 5.9 Individual Muscle and Passive Tissue Forces During Full Flexion and in a More Neutral Lumbar Posture Demonstrating the Shift from Muscle to Passive Tissue The extensor moment with full lumbar flexion was 1 7 1 Nm producing 3145 N of compression and 954 N of anterior shear. The more neutral posture of 1 70 Nm produced 3490 N of compression and 269 N of shear. Fully flexed Neutral lumbar spine lumbar spine Force (N) Force (N) Muscle R rectus abdominis 16 39 L rectus abdominis 16 62 R external oblique 1 10 68 L external oblique 1 10 40 R external oblique 2 7 62 L external oblique 2 7 31 R internal oblique 1 35 1 30 L i nternal oblique 1 35 1 02 R internal oblique 2 29 88 L internal oblique 2 29 1 1 6 R pars lumborum (L 1) 21 253 L pars lumborum (L 1) 21 285 R pars lumborum (L2) 27 281 L pars lumborum (L2) 27 317 R pars lumborum (L3) 31 327 L pars lumborum (L3) 31 333 R pars lumborum (L4) 32 402 L pars lumborum (L4) 32 355 R il iocostalis lumborum 58 1 00 L il iocostalis lumborum 58 1 37 (continued) 1 21 Table 5.9 (continued) Fully flexed Neutral lumbar spine lumbar spine - Force (N) Force (N) M uscle (continued) R longissimus thoracis 93 1 35 L longissimus thoracis 93 1 79 R quadratus lumborum 25 1 55 L quadratus l um borum 25 1 94 R latissimus dorsi (L5) 15 1 01 L latissimus dorsi (L5) 15 115 R m u ltifidus 1 28 80 L m ultifidus 1 28 1 02 R multifidus 2 28 87 L multifidus 2 28 90 R psoas (L 1) 25 61 L psoas (L 1) 25 69 R psoas (L2) 25 62 L psoas (L2) 25 69 R psoas (L3) 25 62 L psoas (L3) 25 69 R psoas (L4) 25 61 L psoas (L4) 25 69 Liga ment Anterior longitudinal 0 0 Posterior longitudinal 86 0 Ligamentum flavum 21 3 ' 22 Normal and Injury Mechanics of the Lumbar Spine 1 23 Table 5.9 (continued) Fully flexed Neutral lumbar spine lumbar spine Force (N) Force (N) Ligament (continued) R i ntertransverse 14 0 L intertransverse 14 0 R articular 74 0 L articular 74 0 R articular 2 1 03 0 L articular 2 1 03 0 I nterspinous 1 301 0 I

nterspinous 2 345 0 I nterspinous 3 298 0 Supraspinous 592 0 R lumbodorsal fascia 1 22 0 L lumbodorsal fascia 1 22 0 demonstrates that the spine is at much greater risk of sustaining shear injury (> 1 000 N applied to the joint) (224 lb) than compressive injUly (3000 N applied to the joint) (672 lb) simply because the spine is fully flexed. (For a more comprehensive discussion, see McGill, 1 997.) The margin of safety is much larger in the compressive mode than in the shear mode since the spine can safely tolerate well over 10 k N i n compression, but 1 000 N of shear causes inju ry with cyclic loading. This example also illustrates the need for clinicians/ergonomists to consider other loading modes in addition to simple compression. In this example the real risk is anterior/posterior shear load. I nterestingly, Norman and colleagues' 1 998 study showed joint shear to be very important as a metric for risk of injury of auto plant workers, particularly cumulative shear (high repetitions of subfailure shear loads) over a workday. Yet another consideration impinges on the interpretation of injury risk. The ability of the spine to bear load is a function of the curvature of the spine in vivo. For example, Adams and colleagues (1994) suggested that a fully flexed spine is weaker 124 Low Back Disorders than one that is moderately flexed. In a recent study, Gunning, Callaghan, and McGill (2001), showed that a fully flexed spine (using a controlled porcine spine model) is 20-40% weaker than if it were in a neutral posture.

-Stoop Versus Squat So much has been written on the stoop style versus the squat style of lifting. Typically, conclusions were based on very simple analyses that measured only the low back moment. The reaction moment is a function of the size and position of the load in the hands and the position of the center of mass of the upper body. As the previous example demonstrated, the issue is actually much more complexthe lumbar spine curvature determines the sharing of the load between the muscles and the passive tissues. In addition, each individual elects the way in which agonists and antagonists are coactivated. Thus, the spine kinematic motion patterns, together with the muscle activation patterns, heavily influence the resulting spine load and the ability of the spine to bear load without damage. The risk of injury is the real issue that motivated most of these types of analyses, yet it was not addressed with sufficient detail as to reach a correct conclusion. Motor Control Errors and Picking Up Pencils Although clinicians often hear patients report injuries from seemingly benign tasks (such as when picking up a pencil from the floor), this phenomenon will not be found in the scientific literature. Because such an injlllY would not be deemed compensable in many jurisdictions, medical personnel rarely record this event. Instead they attribute Figure 5.1 4 A group of power lifters lifted very heavy weights while their lumbar vertebrae motion patterns were quantified. Each joint was skillfully controlled to flex but not fully flex. Each joint was 2-30 away from the fully flexed, calibrated angle (see figure 5.1 5). the cause elsewhere. Moreover, while injury from large exertions is understandable, explaining injlllY that occurs when performing such light tasks is not. The following is worth conSidering. A number of years ago, using video fluoroscopy for a sagittal view of the lumbar spine, we investigated the mechanics of power lifters' spines while they lifted extremely heavy loads (Cholewicki and McGill, Figure 5.1 5 This fluoroscopy image of the power lifter's lumbar spine shows how individual lumbar joint motion can be quantified in vivo. While recording, an injury occurred in one lifter when the vertebra at just one joint went to its full flexion angle and surpassed it by about one-half degree. 1992). The range of motion of the lifters' spines was calibrated and normalized to full flexion by first asking them to flex at the waist and support the upper body against gravity with no load in the hands. During the lifts, although the lifters appeared outwardly to have a very flexed spine, in fact, the lumbar joints were 2-30 per joint from full flexion (see figures 5.14 and 5.15). This explains how they could lift such magnificent loads (up to 210 kg, or approximately 462 lb) without sustaining the injuries

that are suspected to be linked with full lumbar flexion. Normal and Injury Mechanics of the Lumbar Spine 1 2 5 However, during the execution of a lift, one lifter reported discomfort and pain. Upon examination of the video fl uoroscopy records, one of the l umbar joints (specifically, the L2-L3 joint) reached the full flexion calibrated angle, while all other joints maintained their static position (2-3° short of full flexion). The spine buckled and caused injury (see figure 5.16). This is the first observation we know of reported in the scientific l iterature documenting proportionately more rotation occurring at a single lumbar joint; this unique occurrence appears to have been due to an inappropriate sequencing of muscle forces (or a temporary loss of motor control wisdom). This motivated the work of my colleague and former graduate student Professor Jacek Cholewicki to investigate and continuously quantify the stability of the lumbar spine throughout a wide variety of loading tasks (Cholewicki and McGill, 1 996). Generally speaking, the occurrence of a motor control error that results in a temporaty reduction in activation to one of the intersegmental muscles (perhaps, for example, a lamina of longissimus, iliocostalis, or multifidus) may allow rotation at just a single joint to the point at which passive or other tissues could become irritated or injured. Cholewicki noted that the risk of such an event was greatest when high forces were developed by the large muscles and low forces by the small intersegmental muscles (a possibility with our power lifters) or when all muscle forces were low such as during a low-level exertion. Thus, injLIly from quite low intensity bending is possible . Adams and Dolan (1995) noted that passive tissues begin to experience damage w ith bending moments of 60 Nm. This can occur simply from a temporary loss of m uscular support when bending over. This mechanism of motor control error resulting in temporary inappropriate neural activation explains how injLIlY might occur during extremely low load situations, for example, picking up a pencil from the floor fol lowing a long day at work performing a vety demanding job. Figure 5.1 6 The power lifter flexed to grab the weight bar (a) and began to extend the spine during the lift (b). As the weight was a few inches from the floor, a single j oint (L2-L3) flexed to the full flexion angle, a nd the spine buckled (e), indicated by the arrow. 126 Low Back Disorders Sources of Motor Control Errors Another issue must be considered when dealing with the possibility of buckling that results from specific motor control errors. In our clinical testing we observed similarly inappropriate motor patterns in some men who were challenged by holding a load in the hands while breathing 1 0% CO2 to elevate breathing. (Challenged breathing causes some of the spine-supporting musculature to drop to inappropriately low levels in some people; see McGill, Sharratt, and Seguin, 1995.) These deficient motor control mechanisms will heighten biomechanical susceptibility to injury or reinjLIIY (Cholewicki and McGill, 1996). My lab is currently involved in a large-scale longitudinal investigation to assess the power of these motor control parameters together with some personal performance variables and their role in causing back injLI1Y over a multiyear period. Sacroiliac Pain-Is It From the Joint? Bogduk and colleagues (1996) proved that some low back pain is from the sacroiliac joint itself. The following discussion offers other considerations. The work that many have reported over recent years has demonstrated the extraordinaty magnitudes of forces within the torso extensor musculature even during nonstrenuous tasks. While these forces have been interpreted for their mechanical role, clinicians have expressed interest in their potential to cause injury. One possibility worth conSidering is that the high muscular forces may damage the bony attachments of the corresponding muscle tendons. Such damage has perhaps been wrongfully attributed to alternate mechanisms. One such example follows. Pain in the sacroiliac region is common and often attributed to disorders of the sarcoiliac (S1) joint itself or the iliolumbar ligament. For this reason, the role of the musculature may have been

neglected. It is known that a large proportion of the extensor musculature obtains its origin in the SI and posterior superior iliac spine (PSIS) region (Bogduk, 1980). The area of tendon-periosteum attachment and extensor aponeurosis is relatively small in relation to the volume of muscle in series with the tendon complex. From thiS, a hypothesis evolved that the seeming mismatch of large muscle tissue to small attachment area for connective tissue places the connective tissue at high risk of sustaining microfailure, resulting in pain (McGill, 1987). Knowledge of the collective muscle force time histories enables speculation about one-time failure loads and cumulative trauma. For example, if the forces of muscles that originate in the SI region are tallied for the trial illustrated in table 5.4 (page 93), then the total force transmitted to the SI region during peak load would exceed 5.6 kN. Such a load would lift a small car off the ground! The failure tolerance of these connective tissues is not known, which makes speCLIlation over the potential for microfailure difficult. No doubt the risk of damage must increase with the increase in the extremely large loads observed in the extensor musculature and with the frequency of application. Industrial tasks comparable to lifting three containers in excess of 18 kg (40 lb) per minute over an eight-hour day are not unusual, suggesting that the potential for cumulative trauma is significant. This mechanical explanation may account for local tenderness on palpation associated with many of the SI syndrome cases. As well, muscle strain and spasm often accompany SI pain. Nonetheless, treatment is often directed toward the articular joint despite the extreme difficulty in diagnosing the joint as the primaly source of pain. While reduction of spasm through conventional techniques would reduce the sustained load on the damaged connective fibers, patients should be counseled on techniques to reduce internal muscle loads through effective lifting mechanics. This is a Normal and Injury Mechanics of the Lumbar Spine 1 27 single example, of which there may be several, in which knowledge of individual muscle force time histories would suggest a mechanism for injury for which a specificand possibly atypical-treatment modality could be prescribed. Bed Rest and Back Pain While bed rest has fallen from favor as therapy, given the generally poor patient outcome, the mechanism for poor outcome is just now starting to be understood. As noted on page 1 1 3, bed rest reduces the applied load (hydrostatic) below the disc osmotic pressure, resulting in a net inflow of fluid (McGill, van Wijk et al., 1996). McGill and Axler (996) documented the growth in spine length over the usual 8 hours of bed rest and then over continued bed rest for another 32 hours. This is unusual sustained pressure and is suspected to cause backache. Oganov and colleagues (991) documented increased mineral density in the vertebrae following prolonged space travel (weightlessness); this is notable because other bones lost mineral density. It indicates that the spine was stimulated to lay down bone in response to higher loads; in this case, the higher load was due to the swollen discs. The analog on eal1h is lying in bed for periods longer than eight hours-it actually stresses the spinel Moderation in All Th The phrase "moderation in all things" appears to encapsulate a lot of spine biomechanical wisdom; varying loading through constant change in activity is j ustifiable. Obviously, overload is problematic, but the previous example shows that even lying in bed too long is problematic-it increases annulus, end-plate, and veltebral stress! Bending and Other Spine Motion While some researchers have produced disc herniations under controlled conditions (e.g., Gordon et al., 1991), Callaghan and McGill (2001a) have been able to consistently produce disc herniations by mimicking the spine motion and load patterns seen in workers. SpeCifically, only a velY modest amount of spine compression force seems to be required (only 800-1 000 N, or 177-220 lb), but the spine specimen must be repeatedly flexed, mimicking repeated torso/spine flexion from continual bending to a fully flexed posture (see page 1 19). In these experiments, we documented the progressive

tracking of disc nucleus material traveling posteriorly through the annulus of the disc with final sequestration of the nucleus material. This was noted to occur around 18,000-25,000 cycles of flexion with low levels of spine compression (about 1 000 N, or 220 Ib), but it would occur with much fewer cycles of bending with higher simultaneous compressive loads (about 5000 cycles with 3000 N, or 670 Ib, of compression). Most interesting was the fact that the tracking of nuclear material initiated from the inside and slowly worked radially outward, suggesting that, had this been a living worker, there may have been little indication of cumulative damage until the culminating event. Repeated Spine Flexion While much ergonomic effort has been on reducing spine loads, it is becoming clearer that repeated spine flexion-even in the absence of moderate load-will lead to discogenic troubles. Ergonomic guidelines will be more effective once factors for flexion repetition are included. 128 Low Back Disorders Confusion Over Twisting While twisting has been named in several studies as a risk factor for low back injury, the literature does not make the distinction between the kinematic variable of twisting and the kinetic variable of generating twisting torque. While many epidemiological surveillance studies link a higher risk of LBO with twisting, twisting with low twist moment demands results in lower muscle activity and lower spine load (McGill, 1991). Further, passive tissue loading is not substantial u ntil the end of the twist range of motion (Duncan and Ahmed, 1 991). However, developing twisting moment places very large compressive loads on the spine because of the enormous coactivation of the spine musculature (McGill, 1 991). This can also occur when the spine is not twisted but in a neutral posture in which the ability to tolerate loads is higher. Either single variable (the kinematic act of twisting or generating the kinetic variable of twist torque while not twisting) seems less dangerous than epidemiological surveys suggest. However, elevated risk from very high tissue loading may occur when the spine is fully twisted and there is a need to generate high twisting torque (McGill, 1 991). Several studies, then, have suggested that compression on the lumbar spine is not the sole risk factor. Both laboratory tissue tests and field surveillance suggest that shear loading of the spine, together with large twists coupled with twisting torque, increases the risk of tissue injury. Is Viral Involvement Possible in Low Back Pain? Research has suggested that the incidence of some musculoskeletal disorders of the upper extremity are elevated following exposure to a viral infection. Could viral infections also be responsible for low back disorders? While this scenario is possible in individual cases, it could only account for some. Since animal model studies use animals that are screened for disease, with only approved specimens being selected for testing, controlled mechanical loading would seem to account for the significant findings. Viral infections have been linked to an increased probability of developing such diseases as carpal tunnel syndrome or althritis (Mody and Cassim, 1 997; Phillips, 1 997; Samii et al. , 1 996). However, Rossignol and colleagues (1997) found that workrelated factors accounted for the majority of the causes attributed to developing carpal tunnel syndrome. Future research should examine the links between viral infections leading to spinal tissue degeneration and low back injury etiology. They seem to be involved in some select cases. Section Summary From this description of injury mechanisms with several modulating factors, it is clear that multifactorial links exist. Interpretation of these links will enhance injury prevention and rehabilitation efforts. In fact, effective interventions will not occur without an understanding of how the spine works and how it becomes injured. Biomechanical and Physiological Changes Following Injury While the foundation for good clinical practice requires an understanding of the mechanism for injLIIY, an understanding of the lingering consequences is also helpful. Normal and Injury Mechanics of the Lumbar Spine 1 29 Tissue Damage Pathogenesis, Pain, and Performance Many discuss spine injury

as though it is a static entity, that is, by focusing on specific tissue damage. However, since tissue damage causes changes to the joint biomechanics, other tissues will be affected and drawn into the clinical picture. Understanding the links in this issue begins with the concept of pain. Tissue damage causes pain. Some have said there is no proof that this statement is true since pain is a perception and no instrument can measure it directly. This notion ignores the large body of pain literature (a review of which is well outside the scope of this chapter), which has motivated a recent proposal from a diverse group of pain specialists to classify pain by mechanism-specifically, transient pain (which does not produce long-term sequelae) and tissue injury and nervous system injury pain, both of which have complex organic mechanisms (Woolf et al., 1 998). Siddall and Cousins (1997) wonderfully summarized the great advances made in the understanding of the neurobiology of pain-in particular, the long-term changes from noxious stimulation known as central sensitization. Briefly, tissue damage directly affects the response of the nociceptor to further stimulation; over longer periods of time, both increase the magnitude of the response, sensitivity to the stimulus, and size of the region served by the same afferents. Some clinicians have used this as a rationale for the prescription of analgesics early in acute LBP. Furthermore, some have based arguments on whether a certain tissue could initiate pain from the presence or absence of nociceptors or free nerve endings, requiring proof of damage to the specific tissue in question. But tissue damage (along a continuum from cell damage to macrostructural failure) changes the biomechanics of the spine. Not only may this change cause pain, but it appears to initiate a cascade of change that can cause disruptions to the joint and continual pain for years in leading to conditions such as facet arthritis, accelerated annular degeneration, and nerve root irritation, to name a few. (See Kirkaldy-Willis and Burton, 1 992, for an excellent review of the cascade of spine degeneration initiated by damage.) Butler and colleagues (1990) documented that disc damage nearly always occurs before facet arthritis is observed. Injury and tissue damage initiate joint instabilities, causing the body to respond with arthritic activity to finally stabilize. This results in a loss of range of motion and, no doubt, pain (Brinckmann, 1985). Injury Process: Motor Changes It is conclusive that patients reporting debilitating low back pain suffer simultaneous changes in their motor control systems. Recognizing these changes is important since they affect the stabilizing system. Richardson and coworkers (1999) produced quite a comprehenSive review of this literature and made a case for targeting specific muscle groups during rehabilitation. Specifically, their objective is to reeducate faulty motor control patterns postinjury. The challenge is to train the stabiliZing system during steady-state activities and also during rapid voluntary motions and to withstand sudden surprise loads. Among the wide variety of motor changes researchers have documented, they have paid particular attention to the transverse abdominis and multifidus muscles. For example, during anticipatory movements such as sudden shoulder flexion movements, the onset of transverse abdominis has been shown to be delayed in those with back troubles (Hodges and Richardson, 1996; Richardson et al., 1999), Such delayed onset 130 Low Back Disorders of specific torso muscles during sudden events (Hodges and Richardson, 1996, 1999) would impair the ability of the spine to achieve protective stability during a situation such as a slip or fall. The Queensland group developed a rehabilitation protocol specifically intended to reeducate the motor control system for involvement of the transverse abdominis. A number of laboratories have also documented changes to the multifidus complex. Further, in a very nice study of 108 patients with histories of chronic LBD ranging from 4 months to 20 years, Sihvonen and colleagues (997) noted that 50% had disturbed joint motion and 75% of those with radiating pain had abnormal electromyograms to the medial spine extensor muscles. Interestingly

enough, in the many studies on the multifidus, EMG abnormalities in the more lateral longissimus seem to appear along with those in mulifidus in those with back troubles (Haig et al., 1995). Jorgensen and Nicolaisen (987) associated lower endurance in the spine extensors in general, while Roy and colleagues (995) established faster fatigue rates in the multifidus in those with low back troubles. In addition, changes in torso agonist/ antagonist activity during gait (Arendt-Nielson et al., 1 995) have been documented together with asymmetric extensor muscle output during isokinetic torso extensor efforts (Grabiner, Kah, and Ghazawi, 1 992), which alters spine tissue loading. Further, evidence indicates that the structure of the muscle itself experiences change fol lowing injury or pain episodes. Anatomical changes fol lowing low back injury include asymmetric atrophy in the multifidus (Hides, Richardson, and Jull, 1996) and fiber type changes in multifidus even five years after surgety (Rantanen et al., 1993). Long-term outcome was associated with certain composition characteristics. Specifically, good outcome was associated with normal fiber appearance, while poor outcome was associated with atrophy in the type II fibers and a "moth-eaten" appearance in type I fibers. Moreover, even after symptoms have resolved, Hides and colleagues (996) documented a smaller multifidus and suggested impaired reflexes as a mechanism. This theory appears tenable, given documented evidence of this at other joints, particularly at the knee (Jayson and Dixon, 1 970; Stokes and Young, 1 984). Once again, the clinical emphasis on the multifidus may well be because the bulk of the research has been performed on this muscle. Yet researchers who have examined other muscles have observed similar changes in unilateral atrophy-for example, in the psoas (Dangaria and Naesh, 1 998). This collection of evidence strongly supports exercise prescription that promotes the patterns of muscular cocontraction observed in fit spines and quite powerfully documents patho-neural-mechanical changes associated with chronic LBD. Once again, these changes are lasting years-not 6 to 12 weeks! Other Changes Over the past two years we have extensively tested 72 workers. Of those workers, 26 had a history of back troubles sufficient to result in lost time, 24 had some back troubles but not severe enough to ever lose worktime, and the rest had never had any back complaints. Our subjects began by filling out detailed questionnaires that requested information about demographics, physical demands at work, job satisfaction and personal control, work organization, perceived fitness, and chronicity potential. This was followed with approximately four hours of lab testing. This included comprehensive testing for fitness parameters (body mass index, general strength, flexibility, VOzmax, etc.) and a large inventoty of spine-specific parameters (strength, muscle endurance, three-dimensional coupled motion, muscle usage during tasks requiring static balance, unexpected loading, anticipatory reaction, challenged breathing, unrestricted lifting from the floor, etc.). Throughout all tests, subjects were connected to instruments to document three-dimensional lumbar spine motion C3-SPACE, Polhemus, Colchester, Vermont) and torso musculature activity 04 channels of surface EMG). Normal and Injury Mechanics of the Lumbar Spine 1 3 1 Our findings are summarized here: • Having a history of low back troubles was associated with a larger waist girth, a greater chronicity potential as predicted from psychosocial questionnaires, and perturbed flexion to extension strength and endurance ratios, among other factors. • Those who had a histoty of back troubles had a lack of muscle endurancespecifically, a balance of endurance among torso muscle groups. Absolute torso strength was not related, although the ratio of flexor to extensor strength was important. • Those with a history of low back trouble had diminished hip extension and internal rotation, suggesting psoas involvement. • Those with a history of back troubles had a wide variety of motor control deficits including during challenged breathing (as would occur during

challenging work), balancing, having to endure surprise loading, and so on. Generally, those who were poor in one motor task were likely to be poor in another. Given that those workers who had missed work because of back troubles were measured 261 weeks, on average, after their last disabling episode, multiple deficits appear to remain for very lengthy periods. The broad implication of this work is that having a history of low back troubles, even when a substantial amount of time has elapsed, is associated with a variety of lingering deficits that would require a multidisciplinaty intervention approach to diminish their presence. Whether these deficits were a cause of the troubles (perhaps they could have existed prior to the symptoms) or a consequence of the troubles remains to be seen. A longitudinal study based on these variables is under way. References Adams, M., and Dolan, P. (1995) Recent advances in lumbar spinal mechanics and their clinical significance. Clinical Biomechanics, 10 (1): 3. Adams, M.A., Dolan, P. , and Hutton, W.e. (1987) Diurnal variations in the stresses on the lumbar spine. Spine, 12 (2): 1 30. Adams, M.A., and Hutton, w.e. (1985) Gradual disc prolapse. Spine, 1 0: 524. Adams, M.A., and Hutton, w.e. (1988) Mechanics of the interveltebral disc. 1n: Ghosh, P. (Ed.), Tbe biology q/the intervertebral disc. Boca Raton, FL: CRC Press. Adams, M.A., McNally, D.S., Chinn, H., and Dolan, P. (1994) Posture and the compressive strength of the lumbar spine. Clinical Biomechanics, 9: 5-14. Aggrawa II , N.D., Kavr, R., Kumar, S., and Mathur, D.N. (1979) A study of changes in the spine in weightlifters and other athletes. British jou rnal q/Sports Medicine, 13: 58-6 1. Arendt-Nielson, L., Graven-Neilson, T. , Svarrer, H., and Svensson, P. (1995) The influence of low back pain on muscle activity and coordination during gait. Pain, 64: 231 -240. Ashton-Miller,].A., and Schultz, A.B. (1988) Biomechanics of the human spine and trunk. In: Pandolf, K.B. (Ed.), Exercise and sport science reviews (Vol . 16), American College of Sports Medicine Series. New York: Macmillan. Axler, e., and McGill, S.M. (1997). Low back loads over a variety of abdominal exercises: Searching for tbe safest abdominal challenge. Medicine and Science in Sports and Exercise, 29 (6): 804-811. Bearn,].G. (961) The significance of the activity of the abdominal muscles in weigbt lifting. Acta A natornica, 45: 83. Berkson, M.H., Nachemson, A.L., and Shultz, A.B. (979). Mechanical properties of human lumbar spine motion segments. Part II: Responses in compression and shear: Influence of gross morphology. journal o/Biomechanical Engineering, 101 : 53. Bogduk, N. (980) A reappraisal of tbe anatomy of the human lumbar erector spinae. jo1trnal q/ A natomy, 131 (3): 525. 132 Low Back Disorders Bogduk, N., Derby, R., April!, c., Louis, S., and Schwartzer, R. (996) Precision diagnosis in spinal pain. In: Campbell, J (Ed.). Pain 1996-An updated view (pp. 3 13-323) Seattle: IASP Press. Bogduk, N., and Macintosh, JE. (984) The applied anatomy of the thoracolumbar fascia. Spine, 9: 1 64. Brinckmann, P. (985) Pathology of the vertebral column. Ergonomics, 28: 235-244. Brinckmann, P. , Biggemann, M., and Hilweg, D. (988) Prediction of tlle compressive strength of human lumbar vertebrae. Clinical Biomechanics, 4 (Suppl. 2). Buckwalter, JA. (995) Spine update: Ageing and degeneration of the human intervertebral disc. Spine, 20: 1 307-1314. Burnett, A.F., Khangure, M., Elliot, B.C., Foster, D.H., Marshall, R.N., and Hardcastle, P. (1996) Thoracolumbar disc degeneration in young fast bowlers in cricket. A follow-up study. Clinical Biomechanics, 11: 305-310. Butler, D., Trafimow, JH., Andersson, G .B.]., McNiell, T.W., and Hackman, M.S. (1990) Discs degenerate before facets. Spine, 15: 111- 1 13. Carr, D., Gilbertson, L., Frymeyer, J, Krag, M., and Pope, M. (985) Lumbar paraspinal compartment syndrome: A case report with physiologic and anatomic studies. Spine, 10: 816. Callaghan, J.P., Gunning, JL., and McGill, S.M. (998) Relationship between lumbar spine load and muscle activity during extensor exercises. Physical Therapy, 78 (1): 8-18. Callaghan, J P. , and McGill, S.M. (2001a) Intervertebral disc herniation: Studies on a porcine model exposed to highly

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the rod is loaded as before, the rod can sustain the compressive forces successfully. If you reduce the tension in just one of the wires, the rod will buckle at a reduced load; we could actually predict the node, or locus, of the buckle. Compressive loading similar to this has been performed on human lumbar spines. Typically, an osteoligamentous lumbar spine from a cadaver with muscles removed (and no guy wires) will buckle under approximately 90 N (about 20 lb) of compressive load (first noted by Lucas and Bresler, 1961). This is all that a spine can withstand! This analogy demonstrates the critical role of the muscles (the guy wires) to first ensure sufficient stability of the spine so that it is prepared to withstand loading and sustain postures and movement. Also demonstrated with this example is the role of the motor control system, which ensures that the tensions in the cables are proportional so as to not create a nodal point where buckling will occur. Revisiting the buckling injury that we observed fluoroscopically in the power lifter (page 125), we would hypothesize that it was caused by a motor control error in which possibly one muscle reduced its activation or, from the previous analogy, lost its stiffness. The synchrony of balanced stiffness produced by the motor control system is absolutely critical. Now we can address how stability is quantified and modulated. Figure 6.1 The spine is analogous to a fishing rod placed upright with the butt on the ground. When compressive load is applied downward to the tip, it will buckle quickly (a). Attaching guy wires at different levels and in different directions and, most important, tensioning each guy wire to the same tension will ensure stability even with massive compressive loads (b). Note that the guy wires need not have high tension forces but that the tensile forces must be of roughly equal magnitude. This is the role of the musculature in ensuring sufficient spine stability. Lumbar Spine Stability: Myths and Realities 139 Quantitative Foundation of Stability a b c d This section quantifies the notion of stability from a spine perspective. During the 1980s, Professor Anders Bergmark of Sweden very elegantly formalized stability in a spine model with joint stiffness and 40 muscles (Bergmark, 1987). In this classic work he was able to represent mathematically the concepts of "energy wells," stiffness, stability, and instability. For the most part, this seminal work went unrecognized largely because the engineers who understood the mechanics did not have the biological/clinical perspective and the clinicians were hindered in the interpretation and implications of the engineering mechanics. This section synthesizes Bergmark's pioneering effort and its continued evolution in the work of several others and attempts to encapsulate the critical notions without mathematical complexity. If you are mathematically inclined, see Bergmark's original work or its formalization by Cholewicki and McGill (1996). Potential Energy as a Function of Height The concept of stability begins with potential energy, which for our purposes here is of two basic forms. In the first form, objects have potential energy (PE) by virtue of their height above a datum. PE = mass' gravity' height Critical to measuring stability are the notions of energy wells and minimum potential energy. A ball in a bowl is considered stable because if a force (or a perturbation) were applied to it, it would rise up the side of the bowl but then come to rest again in the position of least potential energy at the bottom of the bowl (the energy well) (see figure Φ Q Φ 0 Φ 6.2, a-d). As noted by Bergmark, "stable equilibrium prevails when the potential energy of the system is minimum." The system is made more stable by deepening the bowl and/or by increasing the steepness of the sides of the bowl (see figure 6.3). Thus, the notion of stability encompasses PE = m* g* h Slope = joint stiffness Width = joint laxity Figure 6.2 The continuum of stability. The deepest bowl (a) is most stable, and the hump (d) is least stable.The ball in the bowl seeks the energy well or position of minimum potential energy (m· g. h). Deepening the bowl or increasing the steepness of the sides increases the ability to survive perturbation. This increases stability. Figure 6.3 The steepness of the sides of the bowl corresponds to the

stiffness of the passive tissues of the joint, which create the mechanical stop to motion. The width of the bottom of the bowl corresponds to joint laxity. For example, a positive "drawer test" on the knee would be represented by a flattened bottom of the curve in which small applied forces produce large unopposed motion. 140 Low Back Disorders a the unperturbed energy state of a system and a study of the system following perturbation-if the joules of work done by the perturbation are less than the joules of potential energy inherent to the system, then the system will remain stable (i.e., the ball will not roll out of the bowl). The corollary is that the mechanical system will become unstable and possibly collapse if the applied load exceeds a critical value (determined by potential energy and stiffness). The previous ball analogy is a two-dimensional example. This would be analogous to a hinged skeletal joint that has the capacity only for flexion/extension. Spinal joints can rotate in three planes and translate along three axes, requiring a six-dimensional bowl for each joint. Mathematics enables us to examine a 36-dimensional bowl (6 lumbar joints with 6 degrees of freedom each) representing the whole lumbar spine. If the height of the bowl were decreased in any one of these 36 dimensions, the ball could roll out. In clinical terms, a single muscle having an inappropriate force (and thus stiffness) or a damaged passive tissue that has lost stiffness can cause instability that is both predictable and quantifiable. Some clinicians have confused spine stability with whole body balance and stability, which involves the center of mass and base of support in the context of falling over; this is quite different from spine stability. Figure 6.4, a and b illustrate the mechanics of whole body balance and stability. Figure 6.5, a and b show the clinical practice of prodding patients. This approach is misguided for enhancing spine stability. 6 .6 r--_,- P Figure 6.4 Another type of stability, often confused with spine column stability, involves the center of mass and base of support in the context of falling over. The triangle in (a) is stable because a small perturbation to its top would not cause it to fall.The size of this stability can be quantified, in part, by the size of the angle theta: as the center of the mass approaches a vertical line drawn from the base of support, it would require a smaller perturbing force to fall, demonstrating that it is stable but less so (b). Lumbar Spine Stability: Myths and Realities 141 Figure 6.S Misunderstanding stability, illustrated in the example given in figure 6.4, has led clinicians to try to enhance spine stability by prodding patients and attempting to knock them off balance (a) and (b). This is not spine stability but whole body stability. Potential Energy as a Function of Stiffness and Elastic Energy Storage While potential energy by virtue of height is useful for illustrating the concept, potential energy as a function of stiffness and storage of elastic energy is actually used for musculoskeletal application. Elastic potential energy is calculated from stiffness (k) and deformation (x) in the elastic element, as shown here: p PE = 1/2* k* x2 8:PE-P 8<0 : unstable 8>0: stable PE = 1/2 . k . :x;2 We will use an elastic band as an example. Stretching the band with a stiffness (k) a distance x, it will store energy (PE). The stretched band has the potential to do work when stretched. In other words, the greater the stiffness (k), the greater the steepness of the sides of the bowl (from the previous analogy), and the more stable the structure. Thus, stiffness creates stability (see figure 6.6). More specifically, symmetric stiffness creates Figure 6.6 Increasing the stiffness of the cables (muscles) increases the • stability (or deepens the bowl) and increases the ability to support larger applied loads (P) without falling. But most important, ensuring that the stiffness is balanced on each side enables the column to survive perturbation from either side. Increasing stiffness of just one spring will actually decrease the ability to bear compressive load (lowering PE in one direction and mode). illustrating the need for "balancing" stiffness for a given posture and moment demand. 142 Low Back Disorders p 1 a Figure 6.7 (a) Spine stiffness (and stability) is achieved by a complex interaction of stiffening structures along the

spine and (b) those forming the torso wall. Balancing stiffness on all sides of the spine is more critical to ensuring stability than having high forces on a single side. Load Figure 6.8 If more joules of work are performed on the spine than joules of potential energy due to stiffness, the spine will become unstable-in this case, the instability mode is buckling. even more stability. (Symmetly in stiffness is achieved by virtually all muscles of the torso; see figure 6.7, ab.) If more joules of work are performed on the spine than joules of potential energy due to stiffness, the spine will become unstable (see figure 6.8). Active muscle acts like a stiff spring, and in fact, the greater the activation of the muscle, the greater this stiffness. Hoffer and Andreassen (1981) showed that joint stiffness increases rapidly and nonlinearly with muscle activation such that only very modest levels of muscle activity create sufficiently stiff and stable joints. Furthermore, joints possess inherent joint stiffness as the passive capsules and ligaments contribute stiffness, particularly at the end range of motion. The motor control system is able to control stability of the joints through coordinated muscle coactivation and to a lesser degree by placing joints in positions that modulate passive stiffness contribution. However, a faulty motor control system can lead to inappropriate magnitudes of muscle force and stiffness, allowing a valley for the ball to roll out or, clinically, for a joint to buckle or undergo shear translation. However, we are limited in our ability to analyze the local stability of mechanical systems and particularly musculoskeletal linkages, since the energy wells are not infinitely deep and the many anatomical components contribute force and stiffness in synchrony to create surfaces of potential energy in which many local wells exist. Thus, we locate local minima by examining the derivative of the energy surface. (See Bergmark, 1987; Cholewicki and McGill, 1996, for mathematical detail.) Lumbar Spine Stability: Myths and Realities 143 Spine stability, then, is quantified by forming a matrix in which the total "stiffness energy" for each degree of freedom of joint motion is represented hy a number (or eigenvalue), and the magnitude of that number represents its contribution to for\ning the height of the bowl in that particular dimension. Eigenvalues less than zero indicate the potential for instability. The eigenvector (different from the eigenvalue) can then identify the mode in which the instability occurred, while sensitivity analysis is used to reveal the possible contributors to unstable behavior. Gardner-Morse and colleagues (995) initiated interesting investigations into eigenvectors by predicting patterns of spine deformation due to impaired muscular intersegmental control. Their question was, W hich muscular pattern would have prevented the instability? Crisco and Panjahi (992) began investigations into the contributions of the various passive tissues. Our group has been investigating the eigenvector by systematically adjusting the stiffness of each muscle and assessing stability in a variety of tasks and exercises. The contributions of individual muscles to stability are shown later in this chapter. Activating a group of muscle synergists and antagonists in the optimal way now becomes a critical issue. In clinical terms, the full complement of the stabiliZing musculature must work harmoniously to ensure stability, generation of the required moment, and desired joint movement. But only one muscle with inappropriate activation amplitude may produce instability, or at least unstable behavior could result from inappropriate activation at lower applied loads. Sufficient Stability How much stability is necessary? Obviously, insufficient stiffness renders the joint unstable, but too much stiffness and coactivation imposes massive load penalties on the joints and prevents motion. We can define sufficient stability as the muscular stiffness necessary for stability, with a modest amount of extra stability to form a margin of safety. Interestingly, given the rapid increase in joint stiffness with modest muscle force, large muscular forces are rarely required. Cholewicki's work (Cholewicki and McGill, 1996; Cholewicki, Simons, and Radebold, 2000) demonstrated that, in most people with an undeviated spine, modest

levels of coactivation of the paraspinal and abdominal wall muscles will result in sufficient stability of the lumbar spine. This means that people, from patients to athletes, must be able to maintain sufficient stability in all activities-with low but continuous muscle activation. Thus, maintaining a stability margin of safety when performing tasks, particularly the tasks of daily living, is not compromised hy insufficient strength but probably by insufficient endurance. We are now beginning to understand the mechanistic pathway of those studies showing the efficacy of endurance training for the muscles that stabilize the spine. Having strong abdominals does not necessarily prOVide the prophylactic effect that many hoped for. However, several works suggest that muscular endurance reduces the risk of future back troubles (Biering-Sorensen, 1984). Finally, the Queensland group (e.g., Richardson et al., 1999) and several others (e.g., O'Sullivan, Twomey, and Allison, 1997) noted the disturbances in the motor control system following injury (detailed in chapter 5). These disturbances compromise the ability to maintain sufficient stability. In summary, stability comes from stiffness, passive stiffness is lost with tissue damage, and active stiffness throughout the range of motion is lost with perturbed motor patterns following injuty. Stability Myths, Facts, and Clinical Implications Having explored some of the issues of spine stability, we will now consider a few crucial questions that will help enhance clinical decisions. 144 Low Back Disorders • How much muscle activation is needed to ensure sufficient stability? The amount of muscle activation needed to ensure sufficient stability depends on the task. Generally, for most tasks of daily living, very modest levels of abdominal wall cocontraction (activation of about 10% of MVC or even less) are sufficient. Again, depending on the task, cocontraction with the extensors (this will also include the quadratus) will ensure stability. However, if a joint has lost stiffness because of damage, more cocontraction is needed. A specific example is shown in chapter 13. • Is any single muscle most important? Several clinical groups have suggested focusing on one or two muscles to enhance stability. This would be similar to emphasizing a single guy wire in the fishing rod example. Rarely would it help but rather would be detrimental for achieving the balance in stiffness needed to ensure stability throughout the changing task demands. In particular, clinical groups have emphasized the multifidus and transverse abdominis. The Queensland group performed some of the original work emphasizing these two muscles. This was based on their research, which noted motor disturbances in these muscles following injury. In fact, they developed a tissue damage model that suggested that chronically poor motor control (and motion patterns) initiates microtrauma in tissues that accumulates, leading to symptomatic injury (Richardson et al., 1999). Further, according to the Queensland model, injury leads to further deleterious change in motor patterns such that chronicity can only be broken with specific techniques to reeducate the local muscle-motor control system. The intentions of the Queensland group were to address the documented motor deficits and attempt to reduce the risk of aberrant motor patterns that could lead to the pathology-inducing patterns in their damage model. In other words, their recommendations appear to be directed toward reeducating the faulty motor patterns. However, many clinical groups have interpreted this approach to mean that these two muscles should be the specific targets when teaching stability maintenance over all sorts of tasks. Hopefully the understanding obtained from the stability explanation provided earlier will underscore the folly of this unidimensional emphasis. In fact, the multifidus or any other muscle can be the most important stabilizer at an instance in time. The transverse abdominis, when quantified for its influence on stability, acts the same way. Four exercises are shown as an example (see figure 6.9) where the contributors to stability are shown. You should realize that they will change and adjust with subtle changes in relative muscle activation. In these examples entire muscles are adjusted

to evaluate their role. Obviously, adjustments in single laminae of some muscles would result in yet a different distribution of contributions to stability. Evaluating a wide variety of tasks and exercises is most revealing.The muscular and motor control system must satisfy requirements to sustain postures, create movements, brace against sudden motion or unexpected forces, build pressure, and assist challenged breathing, all the while ensuring sufficient stability. Virtually all muscles play a role in ensuring stability, but their importance at any point in time is determined by the unique combination of the demands just listed. • Do locaVglobal or intrinsic/extrinsic stabilizers exist? From the classic definition and quantification of stability, the answer would be no. Stability results from the stiffness at each joint in a particular degree of freedom. The relative contribution from evelY muscle source is dynamically changing depending on its need to contract for other purposes. The way the various contributors to stiffness add up, however, is important. In some instances, removing a muscle from the analysis has ve,y little effect on joint stability. Once again, it depends on the demands and constraints unique to the task at that instant. The point is that all contributors are important for some tasks and should be recognized as potentially important in any prevention or rehabilitation 8 C <1l<1l .c== ?;'§ 7 xo. <1lc -gg ;'Cii 6 -> ::!i -g", 5 (;) <1lU .c'" _:::J cE 4 ';;;..92 0101 cc 'Cii 3 u'" <1l0l -c ", .- 2 §§ .-to. :::J 0'- o.C 0'" a::E 0 Side bridge Back bridge Sitting on a chair Lumbar Spine Stability: Myths and Realities 145 0.80 0.70 r:::::J Rectus abdominis 0.60 r:::::J External obliques r:::::J Internal obliques 0.50 x <1l Pars lumborum "0 .!: - Iliocostalis lumborum 0.40 - Longisimus thoracis :c '" - Quadratus lumborum 0.30 U5 - Latissimus dorsi - Multifidus 0.20 - Transverse abdominis -+- Stability index 0.10 0.00 Bird dog with arm and leg lift Figure 6.9 Measuring individual muscle influence on lumbar spine stability while performing four different "stabilization" exercises demonstrates that all muscles are important and that their relative importance changes with the task. Total stability is also indicated by the determinant demonstrating how stability can change between tasks. (This is an example from a single subject.Typically, group means indicate a different stability ranking of exercises.) Courtesy of N. Kavcic and S.McGill. program. Conversely, conceiving stabilizers as "intrinsic" or "extrinsic" may offer no benefit for clinical decision making. • What are stabilization exercises? Stability exercises and a stable core are often discussed in exercise forums. What are stabilization exercises? The fact is that any exercise can be a stabilization exercise depending on how it is performed. Sufficient joint stiffness is achieved by creating specific motor patterns. To adapt a phrase popular in motor control circles, "Practice does not make perfect; it makes permanent." Ideally, good stabilization exercises that are performed properly groove motor and motion patterns that ensure stability while satisfying all other demands. Thus, an exercise, repeated in a way that grooves motor patterns and ensures a stable spine, constitutes a stabilization exercise (McGill, 2001). However, some stabilization exercises are better than others; again, it depends on the objectives. For example, the resultant load on the spine is rarely considered. One key to improving bad backs is to select stabilization exercises that impose the lowest load on the damaged spine. Chapter 5 provides a ranking of such exercises. On the other hand, the best stabilization exercise for a high-performance athlete will involve the grooving of a dynamic and complex motion pattern, all the while ensuring sufficient spine stability. An important requirement for many athletes is to ensure a stable spine while breathing hard, such as when playing high-intensity sports. This will be discussed in chapter 14. • Can clinicians identify those who are poor stabilizers? From a qualitative perspective, the Queensland group observed aberrant motor patterns that they hypothesized to compromise the ability of the affected person to stabilize effiCiently. From a quantitative perspective, we have used modeling analysis to try to find ways to identify those who

compromise their lumbar stability from specific motor control errors. We observed such inappropriate muscle sequencing in men who were challenged by holding a load in the hands while breathing 10% COI to elevate breathing. On one hand, the muscles must cocontract to ensure sufficient spine stability, but on 146 Low Back Disorders the other, challenged breathing is often characterized by a rhythmic contraction/relaxation of the abdominal wall (McGill, Sharratt, and Seguin, 1995). Thus, the motor system is presented with a conflict: Should the torso muscles remain active isometrically to maintain spine stability, or will they rhythmically relax and contract to assist with active expiration (but sacrifice spine stability)? Fit motor systems appear to meet the simultaneous breathing and spine support challenge; unfit ones may not. All of these deficient motor control mechanisms will heighten biomechanical susceptibility to injury or reinjury CCholewicki and McGill, 1996). We are currently using this paradigm in a longitudinal study to see if those who bordered on instability during this challenge will be the ones who develop low back troubles. Some other tests to detect functional instability are described in chapter 12. In summary, achieving stability is not just a matter of activating a few targeted muscles, be they the multifidus, transverse abdominis, or any other. Sufficient stability is a moving target that continually changes as a function of the three-dimensional torques needed to support postures. It involves achieving the stiffness needed to endure unexpected loads, preparing for moving quickly, and ensuring sufficient stiffness in any degree of freedom of the joint that may be compromised from injUly. Motor control fitness is essential for achieving the stability target under all possible conditions for performance and injUly avoidance. References Bergmark, A. (1987) Mechanical stability of the human lumbar spine. Doctoral dissertation, Department of Solid Mechanics, Lund University, Sweden. Biering-Sorensen, F. (1984) Physical measurements as risk indicators for low back trouble over a one year period. Spine, 9: 106-119. Cholewicki, J., and McGill, S .M. (996) Mechanical stability of the in vivo lumbar spine: Implications for injUly and chronic low back pain. Clinical Biomecbanics, 11 (1): 1-15. Cholewicki, J., Simons, A.P.D., and Radebold, A. (2000) Effects of external trunk loads on lumbar spine stability. joumal Q/Biomecbanics, 33 (11): 1377-1385. Crisco, J.J., and Panjabi, M.M. (1992) Euler stability of the human ligamentous lumbar spine. Pan I: Theory and Part II: Experiment. Clinical Biomecbanics, 7: 27-32. Gardner-Morse, M., Stokes, I.A.F., and Laible, J.P. (995) Role of the muscles in lumbar spine stability in maximum extension efforts. journal Q/Ol1bopaedic Researcb, 13: 802-808. Hoffer, j., and Andreassen, S. (1981) Regulation of soleus muscle stiffness in prem 0.05 > 1.0 > 0.75 > 4.0 Table 12.1 Mean Endurance T imes (sec) and Ratios Normalized to the Extensor Endurance Test Score Mean age 21 yrs (men: n = 92; women: n = 137) Men Women All Task Mean SO Ratio Mean SO Ratio Mean SO Ratio Extension 161 61 1.0 185 60 1.0 173 62 1.0 Flexion 136 66 0.84 l34 81 0.72 l34 76 0.77 RSB 95 32 0.59 75 32 0.40 83 33 0.48 LSB 99 37 0.61 78 32 0.42 86 36 0.50 Flexion/extension 0.84 0.72 0.77 ratio RSB/LSB ratio 0.96 0.96 0.96 RSB/extension 0.58 0.40 0.48 LSB/extension 0.61 0.42 0.50 RSB = right side bridge; LSB = left side bridge 228 Low Back Disorders Table 12.2 Mean Endurance Times Comparing Normal Workers With Those Who Have Had Back Disorders Men, mean age 34 yr (never had back troubles: n = 24; lost work due to LBO: n = 26) from the same workplace. Variables that are significantly different between those with a history and those who have never had troubles have an asterisk. Note that all men were asymptomatic at the time of testing; these are long-lingering deficits subsequent to back troubles. History of disabling No back troubles back troubles Task Mean SO Ratio Mean SO Ratio Extension 103 35 1.0 90 49 --1.0 - Flexion* 66 23 0.64 84 45 -- --0.93 RSB 54 21 0.52 58 23 0.64 LSB 54 22 0.52 65 27 0.72 - Flexion/extension ratio* 0.71 0.26 1.15 0.66 RSB/LSB ratio* 1.05 0.32 0.93 0.22

RSB/extension* 0.57 0.29 0.97 1.20 LSB/extension* 0.58 0.28 1.03 1.16 Testing for Aberrant Gross Lumbar Motion The usual practice when testing for aberrant gross lumbar motion is to test for range of motion about the three primary axes of motion by determining the degrees of motion possible from neutral (usually using standing posture as a reference base). For example, a flexion range of motion may be determined to be 45° from neutral. As I mentioned earlier, this single number is of little use as an indicator of pathology or for guiding therapeutic exercise decisions. Other well-known markers are the inability to achieve myoelectric silence in the extensors at full flexion or the inability to achieve full flexion (see figure 12.1). However, pathology may manifest within the moderate range of motion (not at end range) and also may occur in axes other than the primary plane of motion. Generally, this pathology is repeatable and evident in each repeated motion cycle. The following examples illustrate this point. The patient in figure 12.2 had a normal range of motion and by traditional tests would be classified as normal. Yet during a lateral bend test (from neutral 0° to 23°), every time the spine bent laterally passing 17°, a small flexion "clunk" (instability) of a degree and a half occurred in the flexion axis. This occurred every time the spine passed 17° of lateral bend. The point is that the motion pathology, or instability, was not in the lateral bend axis (or primalY motion axis) but in the flexion axis (a secondary axis). Clinicians often miss these vely subtle aberrant catches in the "off' axes during examination. The more skilled clinicians sometimes see them or feel them with their hands. :2: O· a: 0 a 0 > :2: 0 b 0 > :2: 0 c 0 > :2: 0 d Flexion Response Test Normal Pathological 3D lumbar 3D lumbar kinematics kinematics 60 60 40 40 20 ..... _-... __ ._---_._-----_.-... 20 0 .' . ' :2: 0 0 -20 - - a: -20 -40 "/ 0 -40 .. --"- -60 -60 _ .. ---_ .. - " -,- -80 -80 " -- - ""-... / -100 e -100 Multifidus Multifidus 100 90 90 80 80 70 70 /,,/- 0 60 60 Silence > 50 50 during full_ .::'.. :2: 40 40 flexion / / 0 30 30 -(20 20 10 10 0 f 0 Lower erector spinae Lower erector spinae 80 70 70 60 60 50 50 0 40 40 > :2: 30 30 0 20 20 10 10 0 9 0 External obliques External obliques 14 14 12 1\ 12 10 10 8 0 8 > 6 :2: 6 4 0 4 2 2 0 h 0 Figure 12.1 Normal 3D motion profiles (a) are shown on the left together with EMG patterns. Myoelectric silence is achieved in the lumbar extensors (b) and (c) during full flexion.This demonstrates the ligaments' and discs' ability to bear the load, suggesting they are in good health. As well, the abdominals are activating (d) to do one of two thingseither the patient is hollowing in the abdominal wall so as not to let it hang, or he is willing to add to the flexor moment to pull into even more full flexion.This too is a marker of good ligament and posterior disc health. The individual on the right cannot fully flex (e) and is unable to achieve lumbar extensor muscle silence (f) and (g). The abdominals are completely unwilling to activate (h), exacerbating the flexion moment. Further testing will reveal troubles in the posterior complex. 229 a b c d 230 Low Back Disorders 6.5 en 3.7 Q) Ol 0.0 Q) 0-2.1 -5.0 0 en -10 Q) Ol Q) 0 -20 -30 0 en -2 Q) Ol Q) -4 0 -6 37 en 25 Q) 14 Ol Q) 0 2 -10 Trial 1 Lateral bend to the right ' , Flexion/extension A Lateral bend Axial twist C Flexion/extension derivative I I I 0 20 40 60 80 100 Figure 12.2 This patient had a normal range of motion but demonstrated an "instability catch." During a lateral bend test (from neutral 0° to 23°), every time the spine bent laterally passing' 7° (event A) (b), a small flexion hitch of a degree and a half occurred in the flexion axis (event B) (a). This occurred very time the spine passed' 7° of lateral bend. There was no deviation in the twist axis (c). The bottom curve represents the first derivative of the flexion curve, which we use to better locate instability. The spike (event C) (d) clearly points to the point at which the instability occurred in the flexion axis. In this patient, a muscle activation pattern was identified that removed the clunk or catch. In figure 12.3, a and b, a "clunk" or catch occurs in the flexion axis as the patient flexes forward and passes 30°; this occurred in evelY flexion attempt. Sometimes patients will report pain as the catch

occurs, and sometimes the clinician notices evidence of muscle activation bursts, but the patient simply moves through the region. We use an electromagnetic instrument that tracks three-dimensional spine motion and graphically displays the results. Sometimes the catches are so subtle that it is far more illuminating to observe the first derivative of the motion axis with the catch. Patients with pain have a much higher incidence of these catches (McGill and Peach, in press) than normals, indicating instability. Normals who demonstrate these catches, although rare, may be at elevated risk for becoming patients according to the aberrant motion model of Richardson and colleagues (999). Testing for Lumbar Joint Shear Stability eha pters 4 and 5 offer a well-developed discussion of the role of the lumbar extensors (longissimus thoracis and iliocostalis lumborum) to support anterior/posterior shear of Evaluating the Patient 231 0; :2- Q) 50 .----------. Cl c 40 30 20 « 10 Flexion Lateral bend Twist ----------._---_ ... _---------- ---- -10+------.------.------.r------r------r------.------.------.------.-----o 2 4 6 8 a 10 Time (s) 12 14 16 18 20 'x ;;::: '0 c o Q) 2 0 .----------. Q) > > -1 .o "2 -2 C') b -3 +------.------.------.------,------.------.-------r------.------.-----o 5 10 15 20 25 Angle (deg) 30 35 40 45 Figure 12.3 In this patient an instability catch or "clunk" occurred in the primary axis of motion (flexion). As he flexed, the clunk was seen in the flexion axis at 30° (event A) (a). This occurred in every trial of forward flexion. The instability is evident (event B) in the third derivative of the flexion axis motion (b). The rehabilitation objective was to stabilize the spine to eliminate the clunk. This was not fully successful over the short term. Symptoms resolved only when the patient developed spine position awareness and was able to avoid flexion to 30°. the vertebral motion segments. These muscles stiffen against unstable motion in the shear plane. In fact, good stabilization exercise must involve these muscles. A simple test can be performed to see if the patient has pain provoked by shear instability and thus could benefit by reducing the aberrant shear motion. If the test is positive, the patient will do well with stabilization exercises that groove cocontracting motor patterns of the shear musculature. D nual Testing for Lumbar Joint Shear Stability The patient lies prone with the body on a table but with the legs over the edge and the feet on the floor.The person must relax the torso musculature.The clinician then applies direct force downward onto each spinous process in turn (a) (starting at the sacrum, then LS, L4, L3, etc.). Unstable segments are identified when either the person reports pain or the clinician feels actual shear displacement, but the patient's reporting of segment-specific pain, in this case, is given more consideration. Then the patient is asked to slightly raise the legs off the floor to contract the back extensors (b). The clinician once again applies force on each spinous process. By virtue of their lines of action, the lumbar (continued) 20 232 Low Back Disorders Manual Testing for Lumbar Joint Shear Stability (continued) a extensors will reduce shearing instability if present. If pain is present in the resting position but then disappears or subsides with the active cocontraction, the test is positive. This test proves conclusively that activating the extensors stabilizes the shear instability and eliminates the pain. Now the trick is to incorporate these extensor motor patterns into exercise prescriptions to carry over to daily activities. A Note on Motion Palpation Some clinicians use their hands to feel specific vertebral motion during a·whole trunk motion such as flexion or twisting. W hen they detect a "blocked" or "stiff-feeling" segment, they often declare it pathological and make it the target of mobilizing treatment. Clinicians rarely, however, consider the flexibility in the spinous process and bony neural arch noted in chapter 4. Two of my PhD students questioned whether the asymmetric feel to a specific joint was true pathology or simply asymmetric anatomyperhaps asymmetric facet joints (Ross, Bereznick, and McGill, 1999). By carefully

documenting the motion occurring at the disc and in the spinous process in cadaveric specimens, together with the applied loads (similar to clinical loads), they were able to quantify the effect of anatomy. The results demonstrated that over half of the motion felt by the clinician was actually the spinous process flexing and bending (see figure 12.4) and not vertebral body motion. Those joints that were anatomically asymmetric had asymmetric motion under an applied load from the clinician. Treating such a joint with mobilization therapy would be fruitless. Testing for Aberrant Motor Patterns During Challenged Breathing The test for aberrant motor patterns during heavy breathing is rather sophisticated, but it is about the only way to identify a persons' ability to maintain spine stability during tasks that require higher physiological work rates. Candidates for this type of test include athletes and occupational workers such as construction workers, warehouse employees, and so on. We explored the link between breathing and spine stability Evaluating the Patient 233 80 70 60 50 z Q) 40 0 u.. 30 20 Actual vertebra motion 10 Spinous process deflection 0 -0.5 0 0.5 1.5 Figure 12.4 Lateral forces were applied to the tip of the spinous process by clinicians to cadaveric motion segments to mimic motion-palpation testing. Most of the motion measured in the spinous process, which is what the clinician feels, took place in the spinous process bending rather than in the disc. Moreover, asymmetric anatomy resulted in asymmetric motion when loaded to mimic the clinician's hand forces, suggesting that this test should not be used solely to determine the site of mobilizing treatment. previously (pages 145-146). Challenged breathing requires heavy involvement of the abdominal muscles. Because we monitor the muscles with EMG electrodes and because muscle fatigue changes the EMG signal, we avoid creating heavy lung ventilation with exercise for this test. (Note that this consideration is only for testing and that we will intentionally increase the physiological work rate for exercises in chapter 14.) DChalienged Breathing Test During this test the person breathes a 10% CO2 gas mixture to elevate the lung ventilation with only a few breaths, but the 02 is adjusted so that the light-headedness that accompanies hyperventilation does not occur.Then a weight, usually about 15 kg (33 Ib) for the average man, is placed in the hands while the torso is flexed forward about the hips to approximately 30°. This test reveals those who have aberrant motor patterns in the abdominals during challenged breathing that compromise lumbar stability. Q) "0 . -E 0.. E <1I "0 Q) . Cii E 0 Z a 234 Low Back Disorders 20 15 10 5 0 -5 -10 Good spine stabilization patterns observed in the muscle EMG signal obtained during the challenged breathing test include a constant muscle cocontraction ensuring spine stability. Figure 12.5 shows stable patterns (a) and unstable patterns (b). Our computerized analysis package measures spine stability using the methods explained in chapter 6. Many clinicians will not have this capability but should still look for constant levels of abdominal wall activation. Patients who are poor at stabilization in these situations will demonstrate short, temporary reductions in activation or even loss of activation. These mark the critical instances at which spine instability can occur and classify the person as a candidate for spine stabilization during high physiological work rates. These patients must learn to contract the spine stabilization muscles independently of challenged breathing to ensure spine stability in conditions of heavy work. Determining Suitability for ROM Training and Stretching The McKenzie extension approach is well-known for treating acute discogenic troubles. The extension postures can also be used as a test to identify those with posterior disc troubles. Typically, these posterior discogenic patients should never flex the lumbar spine when it is under substantial loading. .....•..... ....••.....•.....•.....•.. .... , ...••... J .... , II I il\ I 'I ..... 9.g.J.?t:J.t .rl1.lJ.C.IE3 .. ?t.i.\lClt.i.()fl ..... ; ..... I\ ..... \ .. •.....) ..... !\ ...) ..... · j···· ····· ····· ····· . ····· ..

…….⋯ ..• · .. ;, ... "(".;:; ..... r' .. □ ..... i'\ .... .. j.;\ ....).,; ..... !\ ..... (.□.!.\ .... /\ .. }.□ ... I .. □\ ... j,.□ .... ! . .I .. I) .... ,i.:.□: ... .. .I.!! ..... □ ... : .. 1 •.... , .•.•..• 1 ••.•••..••.•.•...•. .v • •. ;\/.. " . . "□□j . ; \/ .. ' . \ I. '1'" .. i . I 1 ..... r .• I" .•.. :::::: . .. . :::::.:.:: . . :::::.::::: ::. :::::::::::::: :::::.:· · ::·:·:::· ··:: . .. ···· . .. · ::: ::: · :Ji:::· :::· ::: :.: .. :: .. : .. ::.:· j: . :: : ::. · : : · ..... , ... Ventilation I□ f' □ r! t ,J -15 .... -20
+----------.----------,----------.----------,----------,----------□ o 10 20 30 40 50 60 Time 1 20 5 .----------, ... Variable muscle activation 10 ..... . 5 "0 0 Q) .□ Cii -5 E o -10 Z -15 b -20
+----------,----------,----------,----------,----------,----------□ o 10 20 30 40 50 Time Figure 12.5 Good spine stabilization patterns during the CO2 breathing test include a constant muscle cocontraction, which ensures spine stability. Both stable (a) with constant muscle activation and unstable (b) variable abdominal muscle activation patterns are shown. 60 Evaluating the Patient 235 DMcKenzie Posture Test The patient begins in a relaxed standing posture, and the clinician asks him about current pain and the general feeling about the back. Then the patient lies prone, adopting one of the following three levels of the McKenzie posture progression (the clinician decides which is suitable based on the patient's flexibility and symptoms): • Prone with the arms relaxed (a) • Prone with the chin resting on the fists (b) • Prone supported on the elbows (c) If the patient cannot tolerate one of these postures, the clinician can assume that simple disc disturbances are not the source of troubles or at least not the sole source. Many with uncomplicated posterior disc lesions, or herniations, find relief in these postures. The patient then returns to the standing posture. If he feels more stable and/or has less pain compared to the moments prior to lying prone, he is cla ssified as posterior discogenic. Patients who are posterior discogenic should avoid flexion stretches and a flexed spine under load when performing exercise therapy. Those who feel worse upon standing could have a host of other possible conditions but will probably experience better progress by adopting a neutral to slightly flexed lumbar posture for exercise therapy. 236 Low Back Disorders Distinguishing Between Lumbar and Hip Problems Pain in the buttock and/or radiating down the leg can have more than one source. For effective treatment, you must discover whether that source is in the lumbar spine or in the sacroiliac (SI) joints, in various muscles, or in tissues around the hip or even the hip joint itself. g Slump Test Several forms of the sitting slump test can be used to elicit radiating sciatic symptoms. These are designed to tense the sciatic nerve and irritate the lumbar nerve roots. Typically the patient sits on the table or chair and then slumps or slouches. The intention is to progressively increase the nerve tension. Then • the leg can then be extended at the knee (a), tensing the nerve from below the lumbar spine; • foot flexion can be added (b), further tensing the nerve; and finally • the nerve can be further tensed from above the lumbar region with the addition of cervical spine flexion (c). If the tension at any of these stages causes symptoms, further progressive testing is stopped, and the patient may be a candidate for nerve flossing (described in chapter 13), Certainly flexion will be contraindicated for these patients during stretching and exercise. If flexing the neck does not increase the pain, then the sciatic nerve is not the tissue of interest and the clinician can begin assessing the SI joints, the hamstrings, piriformis, and other muscle-based syndromes, ending with the hip. jersztajn Test The Fajersztajn test should always be performed on both legs even when the sciatica is unilateral. Raising the "well leg" is needed to perform the "well leg raising test of Fajersztajn" (DePalma and Rothman, 1970). Raising the well leg tensions the nerve root on

the well side together with causing tensions centrally along the midline of Evaluating the Patient 237 the cauda equina and to the nerve roots on the opposite side (a). Sometimes pain is provoked with simultaneous cervical flexion (b). Pain in the symptomatic side (side not raised) is an o rganic sign of disc lesion, usually a more central lesion. It is not a sign of malingering as some have suggested. I am continually surprised at the number of people with back troubles who also have hip troubles (McGill and colleagues, in press, recently noted a correlation between back troubles and hip pathology in factory workers). It is sometimes difficult to separate hip pain from lumbar trouble since beep buttock pain can be sciatic (lumbar) or originate in the hip joint. Typically, medial and anterior thigh pain are indicators of hip pathology. If hip tests are indicated by the nerve tension tests described in the previous section (j.e., they were negative), flexion and rotation tests should be performed. Dttip Flexion and Rotation Tests Lying supine with the hip flexed, the patient's thigh segment is rotated into hip internal rotation (a). Stiffness or pain is a positive indicator of hip concerns. Hip joint pain is somewhat distinctive with radiation into the groin, and/or along the inguinal crease, and/ or medial/anterior thigh. The patient's hip is then flexed (b) while the knee is moved in circula r motions, scouring the acetabulum but with the knee bent so as not to confuse any pain with nerve tension related to the back. 238 Low Back Disorders References Biering-Sorensen, F. (1984) Physical measurements as risk indicators for low-back trouble over a one-year period. Spine, 9: 106-119. DePalma, A.F. , and Rothman, R.H. (1970) The intervertebral disc. Philadelphia: W.B. Saunders. McGill, S.M., Childs, A., and Liebenson, C. (1999) Endurance times for stabilization exercises: Clinical targets for testing and training from a normal database. Archives of Physical Medicine and Rehabilitation, 80: 941-944. McGill, S.M., Grenier, S., Preuss, R., and Brown, S. (in press) Asymmetries in torso endurance and strength parameters are associated with a histOIY of low back troubles. McGill, S.M., and Peach ,]. (in press) Electromyographic and spine kinematic changes with pain. Richardson, c., lull, G., Hodges, P. , and Hides,]. (1999) Therapeutic exercise for spinal segmental stabilization in low back pain. Edinburgh, Scotland: Churchill Livingstone. Ross,].K., Bereznick, D., and McGill, S.M. (1999) Atlas-axis facet asymmetry: Implications for manual palpation. Spine, 24 (12): 1203-1209. Developing the Exercise Program The exercises described in this chapter comprise a program founded on the principles discussed in previous chapters. This program is sufficient for many patients and for those interested in optimal back health. For those with performance ambitions, chapter 14 offers more advanced versions. While I claim no expertise beyond the low back, I will broach other issues that have implications for the back. For example, improper technique when pelforming hip and knee mobilization and stretching can compromise the back; for this reason I have included them here. Upon completion of this chapter you will be able to design exercise that challenges muscle and establishes stabilizing motor patterns, but spares the spine. The focus here is clearly on the patient and low back health for evelyday activity. Preliminary Matters Before presenting the exercises, I will review some of the basic ideas discussed in the text thus far, establish how to stretch without stressing the back, clarify issues regarding abdominal hollowing and bracing, and introduce the technique of flossing the nerve roots for those with sciatica. Philosophy of Low Back Exercise Design Many traditional notions that exercise professionals consider to be principles for exercise design, particularly when dealing with the low back, may not be as well supported with data as generally thought. As previously noted, many clinicians still prescribe sit-ups-usually with the proviso that the knees remain bent. The resultant spinal loading of well over 3000 N of compression to a fully flexed lumbar spine clearly shows the folly of such a recommendation. Other experts still recommend the posterior pelvic tilt when performing many types of low

back exercise. This actually increases the risk of injury by flexing the lumbar joints and loading passive tissues. The recommendation of flattening the lumbar region to the floor when performing 239 240 Low Back Disorders abdominal exercise is another version of this ill-founded philosophy. Many continue to believe that having stronger back and abdominal m uscles is protective and reduces bad back episodes, even though Luoto and colleagues (995), among others, showed that m uscle endurance, not strength, is more protective. This should not be m isinterpreted to mean that having stronger m uscles is not a good objective; rather, it reflects on m any of the strength training approaches currently used that create back patients. Myths still remain regarding the need for greater lumbar mobility, which the evidence suggests leads to more back troubles-not less (e.g. , BieringSorensen, 1984)! Finally, we are disturbed by the fact that replicating the motion and spine loads that occur during the use of m any low back extensor machines used for training and therapy produced disc herniations when applied to spines in our laboratory! Clinical Wisdom Clearly, some current clinical wisdom needs to be reexamined in the light of the scientific evidence, much of which has been presented in this book. This evidence seems to indicate that the safest and most mechanically justifiable approach to enhancing lumbar stability through exercise is to emphaSize endurance over strength. Clinicians using such an approach should encourage patients to maintain a neutral spine posture when under load and use abdominal cocontraction and bracing in a functional way. The bodybuilding approach often used in rehabilitation is not synonymous with health objectives and results in additional risk to create joint loading through a range of motion and the often simple motor patterns that are not optimal for ensuring joint stability. Sparing the Back While Stretching the Hips and Knees Given that spasms in the psoas, which produce a shortening and hip extension restriction, are common in those with back troubles (Grenier et al. , in press), these people would probably benefit from hip stretching and other range-of-motion exercises. Sometimes, however, poor technique when stretching hip and knee tissues leads to unnecessary loading of the lumbar spine. This can easily be avoided by changing technique. A general guideline for sparing the back is to maintain an upright torso posture while performing hip and knee work. Preservation of a neutral spine ensures minimal loading from passive tissues. An upright torso minimizes the reaction torques, the associated muscle contraction, and spine load. Abdominal bracing is sometimes required for pain control. For example, lunges are a good exercise for challenging strength, endurance, balance, and mobility in the lower extremities. Lower extremity capability is needed to facilitate spine-sparing postures when lifting and when performing a host of other tasks. n .. rlnn the Back The technique for sparing the back is to maintain an upright torso posture while performing the lunge (see figure 13. 1). Some clinicians recommend keeping the back leg straight, which causes patients to flex the torso forward. This is poor form for sparing the back (see figure 13.2). A general principle is to keep the torso upright with a neutral lumbar spine while stretching joints other than the back. Developing the Exercise Program 241 Figure 13.3, for example shows correct postures for stretching the quads (a) and the hip adductors (b). Finally, the lunge with a neutral spine stretches iliacus more than psoas (iliacus is a uniarticular m uscle that crosses only the hip). Full stretch on the psoas requires a lateral bend of the torso away from the extended hip, but this is reserved for the more robust backs (see figure 13.4 a-b). Figure 13.1 The spine is spared performing the lunge by maintaining an upright torso and a neutral spine curvature. Figure 13.2 Some clinicians recommend keeping the back leg straight while performing lunges, which causes patients to flex the torso forward. This is poor form for sparing the back. Figure 13.3 During stretching at joints other than the back, the spine is spared by maintaining the upright torso and neutral spine curve. When stretching the quads,

holding a chair for balance is a good way to ensure a straight back (a). The person shown in (b) has sufficient hip flexion mobility to ensure an upright torso and a neutral spine. Individuals who are unable to maintain this spine posture should forgo this exercise until they can achieve the required hip flexion mobility. (continued) 242 Low Back Disorders Sparing the Back (continued) Figure 13.4 Given the uniarticular iliacus and the multiarticular psoas that crosses the hip and entire lumbar spine, a true psoas stretch requires the torso to be laterally bent away from the extended hip; this is an advanced stretch for the more robust backs. Flossing the Nerve Roots for Those With Accompanying Sciatica Before presenting the flossing technique for sciatica, some background is required regarding the biomechanics and biochemistry of the spinal cord and nerves. Excessive tension in the nerve is the cause of sciatica. Normally, the cord nerve roots travel through the foramenae of the neural arch of each vertebra, and their sliding excursions are substantial with spine flexion, hip flexion, and knee extension. According to Louis (1981), the nerve can travel well over a centimeter (over 1/2 in.) during some of these movements. Thus, if a nerve root is impinged and cannot slide, any of these postures that would normally pull that nerve root through the foramen will increase nerve tension. Instead of moving, the nerve is stretched. Further, tension on such a nerve can be increased from the cranial end with simultaneous cervical flexion because the entire spinal cord moves slightly with cervical flexion and thus pulls at the nerve roots all along its length. This is the basis for the slump test for nerve tension presented in chapter 12. Recently, scientists have suggested that nerves have the ability to create their own pathways as long as they can move. They seem to have some chemically based ability to dissolve-over time-tissues impinging them. The idea of "flossing" is to pull the cord and nerves from one end only while releaSing at the other, and then to switch the pull/release direction. In this way the nerve roots are "flossed" through the lateral Developing the Exercise Program 243 foramenae and in fact along their entire length. Thus, by working the nerves back and forth in whatever limited range they can manage in spite of impingement, we facilitate the dissolving of the impingement and the gradual release of the nerve to once again move freely (this approach was proposed by Butler, 1999). This flossing action is accomplished with coordinated hip, knee, and cervical motion. The patient, seated and with the legs able to swing freely, flexes the cervical spine (a). This creates a pull on the spinal cord from the cranial end and a release from the caudal end. This should not produce sciatic symptoms (see the discussion of the slump test in chapter 12). Then, the patient extends the cervical spine with simultaneous knee extension on the side with sciatica (b). This pulls the nerve from the caudal end with a corresponding release at the cranial end. The cycle is completed as the knee is flexed with coordinated motion of the cervical spine flexing.This causes the nerves to floss through the vertebral tissues. If the patient experiences minor sciatic symptoms when the cervical spine is flexed or the knee is extended, then he should reduce the range of motion at these joints until no pain is provoked. Some patients who have suffered sciatica pain for years report reductions in their sciatic symptoms within a few days to a couple of weeks; others report increasing symptoms. A note of caution is needed here: while this can be wonderful in helping chronic sciatica resolution, it can also cause an acute onset. Be very conservative in the first session. If the nerve is adhered so that it can't slide, the sciatic symptoms will be exacerbated. However, if the patient reports no change or even relief the next day, then proceed to increase the flossing. We have not been able to uncover a procedure that predicts who will do well or who will be exacerbated from this flossing procedure. Monitor all patients and remove the procedure from the programs of any patients whose symptoms worsen. 244 Low Back Disorders Exercises for Training the Stabilizing Muscles After a brief review of the muscles that stabilize the spine, we will have a look at

exercises for training those muscles. What Are the Stabilizers of the Lumbar Torso? While many muscles have been regarded as primaly spine stabilizers, confirmation of their role requires two levels of analysis. First, engineering-stability analysis must be conducted on anatomically robust spine models to document the ability of each component to stiffen and stabilize. Second, electromyographic recordings of all muscles (even deep muscles requiring intramuscular electrodes) are necessary to confirm the extent to which the motor control system involves each muscle to ensure sufficient stability. Our intramuscular and surface EMG and modeling studies to quantify spine stability demonstrated that virtually all torso muscles play a role in stabilization. (Some data was presented in chapter 6.) While multifidus, the other extensors, and the abdominal wall all appear to play significant roles, the quadratus lumborum also appears to play an important role in many tasks. The fibers of quadratus lumborum cross-link the vertebrae, they have a large lateral moment arm via the transverse process attachments, and they traverse between the rib cage and iliac crests. In this way, the quadratus buttresses shear instability and appears to be effective in all loading modes, by its architectural design. Typically, the first mode of lumbar buckling is lateral; the quadratus appears to play a significant role in local lateral buttressing. The three layers of the abdominal wall are also important for stability together with the muscles that attach directly to vertebrae-the multisegmented longissimus and iliocostalis and the unisegmental multifidii. Cholewicki and McGill (996) also presented an argument for the role of the small intertransversarii in producing small but critical stabiliZing forces. On the other hand, psoas activation appears to have little relationship with low back demands; the motor control system activates it when hip flexor moment is required (see Andersson et aI. , 1996, and JukeI' et aI. , 1998), compromising its role to stabilize the spine when hip tlexion is not involved. So which are the wisest ways to challenge and train these identified stabilizers? This question will be answered in the follOWing sections. Training the Stabilizers of the Lumbar Spine: The "Big Three" Quantitative data have confirmed that no single abdominal exercise challenges all of the abdominal musculature while sparing the back (Axler and McGill, 1997). For this reason, more than one single exercise is required. Unfortunately, many inappropriate exercises have often been prescribed for people with low back difficulties, including the following: • Sit-ups (both straight-leg and bent-knee) are characterized by higher psoas activation, with consequent high low back compressive loads that exceed NIOSH occupational guidelines. • Leg raises cause even higher psoas activation and spine compression (actual values were presented in chapter 4). Developing the Exercise Program 245 Upper and Lower Rectus Abdominis? Myoelectric evidence, normalized and calibrated, suggests that there is no functional distinction between an "upper" and "lower" rectus abdominis in most people; in contrast, the obliques are regionally activated with upper and lower neuromuscular compartments as well as medial and lateral components. In our investigations we made several relevant observations regarding psoas activation during abdominal exercises. The challenge to the psoas is lowest during curl-ups, followed by higher levels during the horizontal side bridge. Bent-knee sit-ups were characterized by larger psoas activation than straight-leg sit-ups, and the highest psoas activity was observed during leg raises and hand-on-knee flexor isometric exertions. The often-recommended "press-heels" sit-up, which has been hypothesized to activate Figure 13.5 The press-heels sit-up was proposed by several clinical groups, on theoretical grounds, to inhibit psoas by activating the hamstrings. In fact, electromyographic assessment (Juker et aI., 1998) proved this to be mythical.This original photo from the intramuscular experiments shows the clinicians' hands behind the heels of the subject as they activate hamstrings during the sit-up. Activating the hamstrings creates a hip extensor moment, and

sit-ups require hip flexion. During this type of sit-up, the psoas is activated to even higher levels to overcome the extensor moment from the hamstrings and produce a net flexor moment. This type of sit-up produced the highest level of psoas activation of any style of sit-up we quantified! Training Rectus Abdominis hamstrings and neurally inhibit psoas, was actually confirmed to increase psoas activation! (See figure 13.5.) (Original data can be found in Juker et al.,1998; some clinicians and coaches who intentionally wish to train psoas will find this data informative.) Once again, the horizontal side support appears to have merit as it challenges the lateral obliques and transverse abdominis without high lumbar compressive loading and ensures a high stability index (a loss of contraction of the involved muscles would cause the patient to fall out of the bridge). A wise choice for stabilization exercises in the early stages of training or rehabilitation, and for simple low back health objectives, would be the "big three" that we have quantified to sufficiently challenge muscle, spare the spine of high load, and ensure sufficient stability: 1. Curl-ups for rectus abdominis 2. Several variations of the side bridge for the obliques, transverse abdominis, and quadratus 3. Leg and arm extensions leading to progressions of the "birddog" for the many back extensors The variation of each of these exercises must be chosen with the patient/athlete's status and goals in mind. Calibrated intramuscular and surface EMG evidence suggests that the various types of curl-ups challenge mainly rectus abdominis since psoas and abdominal wall (internal and external oblique and transverse abdominis) activity is relatively low (see tables in chapter 4 for relative activation levels in a variety of exercise tasks). Curl-ups performed with poor technique, however, can be counterproductive, either failing to 246 Low Back Disorders Figure 13.6 Poor form during the curl-up is to flex the cervical spine, loading the neck and not the rectus (a). Correct technique focuses the rotation in the thoracic spine. Another common type of poor form is to elevate the head and shoulders a large distance off the floor (b). This patient is elevating far too much, which is closer to replicating the much higher stresses of the sit-up. The intention is to activate rectus and not to produce lumbar spine motion. activate the rectus abdominis sufficiently or overstressing the spine (see figure 13.6, a and b). Curl-ups with a twisting motion are expensive in terms of lumbar compression due to the additional oblique challenge. Higher oblique activation with lower spine load is accomplished with the side bridge, which is therefore preferred over twisting curl-ups for training the obliques. This will be presented in the next section. The highest-level curl-up will be presented in chapter 14. p, Beginner's The curl-up technique is critical to spare the spine.The basic starting posture is supine with the hands supporting the lumbar region. Do not flatten the back to the floor, which takes the spine out of elastic equilibrium and raises the stresses in the passive tissues. While the position of elastic equilibrium is desired in the lumbar region, the hands can be adjusted to minimize pain if needed. One leg is bent with the knee flexed to 900 while the other leg remains relaxed on the floor. This adds further torque to the pelvis to prevent the lumbar spine from flattening to the floor. The focus of the rotation is in the thoracic spine; many tend to flex the cervical spine, which is poor technique. Rather, picture the head and neck as a rigid block on the thoracic spine. No cervical motion should occur, either chin poking or chin tucking. Individuals who report neck discomfort may try the isometric exercises for the neck that follow. In addition, particularly for patients experiencing neck discomfort, the tongue should be placed on the roof of the mouth behind the front teeth, which helps to promote stabilizing neck muscle patterns. Leave the elbows on the floor while elevating the head and shoulders a short distance off the floor. The rotation is focused in the midthoracic region. The head/neck is locked onto the rib cage. No cervical motion should occureither chin poking or chin tucking. The intention is to activate rectus and not to produce

spine motion. Developing the Exercise Program 247 A very mild form of the curl-up is to just take the weight of the head and shoulders with almost no motion (a), while more challenge is obtained with raising the head and shoulders but focusing the motion to the thoracic spine (b) with no cervical or lumbar flexion. Those who experience neck symptoms with curl-ups may find relief by building the neck with isometric exercises. In all of these exercises the head/neck does not move,and the tongue is placed on the roof of the mouth behind the front teeth: (a) the hands are placed on the forehead, which resists neck flexion effort; (b) the hand is placed on the side of the head to resist cervical side flexion effort, and then repeated on the other side; (c) the hands are placed on the back of the head to resist cervical extension effort. Hold for several seconds then relax, building up endurance and grooving stabilizing motor patterns by increasing the repetitions of the hold/relax cycles. (Believe it or not, some patients try this while chewing gum, which makes the grooving of stabilizing patterns impossible.) 248 Low Back Disorders p,Advanced The intermediate progression of the curl-up is to raise the elbows a couple of centimeters so that the arms do not pry the shoulders up, thus shifting more load to the rectus. Do not raise the head/neck any higher than in the beginner's curl-up. An advanced curl-up requires placing the fingers lightly on the forehead (never behind the head, which causes the tendency to pull and flex the cervical spine), as long as lumbar posture is controlled and no pressure is applied to the head by the hands. Note:The average patient need not go to this level if spine health is the objective. The head and neck must move as a . unit, maintaining their rigid-block position on the thoracic spine. Training the Quadratus Lumborum, Lateral Obliques, and Transverse Abdominis Given the architectural and electromyographic evidence for the quadratus lumborum, transverse abdominis, and abdominal obliques as spine stabilizers, the optimal technique to maximize activation but minimize the spine load appears to be the side bridge. Abdominal bracing is emphasized in all forms of this exercise. Maintaining the bridge ensures constant muscle activation while the brace introduces new combinations of muscle recruitment to ensure stability. It is almost impossible for the spine to become unstable while performing a side bride with a neutral spine. Developing the Exercise Program 249 Many special patient cases deserve consideration-for example, the chronic patient who is quite deconditioned.These patients are sometimes unable to perform the side bridge even from the knees. Start these people with a side bridge while standing against a wall. Instruct them to move smoothly from the beginning position (a) through the intermediate positions (b-d) to the final position (e), in one flowing motion. The patient should pivot over the toes, as if performing a fluid dance move. Once patients graduate to the floor, they can spare the spine by beginning with the knees and hips quite flexed (f) and moving into the side-bridge posture with an accordion-like unfolding of the legs, all the time keeping a braced neutral spine (g). Another type of patient is the football player whose shoulders are so painful he can not tolerate the shoulder load (some elderly women also fall into this patient group). These patients can perform a modified side bridge by lying on the floor and attempting to raise the legs laterally (h and i) or simply attempting to take the weight off the legs. (continued) 250 Low Back Disorders Side Bridge, Remedial (continued) Another option for patients with shoulders that cannot tolerate load is to stand on a 45° bench with the feet anchored U}, which spares the shoulders. Using other devices, the torso can either be elevated from the pad (k and I) or bridged with the shoulder (m). ge, Beginner's ge, Intermediate Developing the Exercise Program 251 Beginners bridge from the knees. In the beginning position, the exerciser is on his side, supported by his elbow and hip. The knees are bent to 900• Placing the free hand on the opposite shoulder and pulling down on it will help stabilize the shoulder. The torso is straightened until the body is supported on the elbow and the knee, with some

input from the lower leg. The beginner's side bridge can be slightly advanced by placing the free arm along the side of the torso-effectively placing more load on the bridge. The beginning position for the intermediate side bridge is like that for the beginner, except that the legs are straight. The torso is straightened until the body is supported on the elbow and feet. (See figure.) When supported in this way, the lumbar compression is a modest 2500 N, but the quadratus closest to the floor appears to be active up to 50% of MVC (this is a preferred exercise for the obliques since they experience similar levels of activation). 252 Low Back Disorders ge, Intermediate Variation Placing the upper leg/foot in front of the lower leg/foot will enable longitudinal"rolling" of the torso to challenge both anterior (a) and posterior (b) portions of the abdominal wall. g&#xfffd;Advanced Advanced technique to enhance the motor challenge of the side bridge is to transfer from one elbow to the other while abdominally bracing (a, b, and c) rather than repeatedly hiking the hips off the floor into the bridge position. Ensure that the rib cage is braced to the pelvis and that this rigidity is maintained through the full roll from one side to the other. Still higher levels of activation would be reached with the feet on a labile surface (Vera-Garcia, Grenier, and McGill, 2000). Training the Back Extensors Developing the Exercise Program 253 Most traditional extensor exercises are characterized by vely high spine loads, which result from externally applied compressive and shear. forces (either from free weights Figure 13.7 A commonly prescribed spine extensor muscle challenge involves lying prone while extending the arms and legs. This results in over 6000 N of compression to a hyperextended spine. It is a poorly designed exercise. or resistance machines). A commonly prescribed spine extensor muscle challenge involves lying prone while extending the arms and legs (see figure 13. 7). This results in over 6000 N of compression to a hyperextended spine, transfers load to the facets, and crushes the interspinous ligament. Needless to say, this exercise is contraindicated for anyone at risk of low back injury-or re-injUly! Although some may believe that putting the hands on either side of the head rather than extending them may make this exercise safe, that is not true. It should be not be done in any form. Further, recall that the mechanism for disc herniation is reproduced by back machines that take the lumbar spine from full flexion and through the range of motion under load from muscle contraction. In our search for methods to activate the extensors (including longissimus, iliocostalis, and multifidii) with minimal spine loading, we have found that the Single leg extension hold results in tolerable spine loading «2500 N) for many and activates one side of the lumbar extensors to approximately 18% of MVC. Simultaneous leg extension with contralateral arm raise (the "birddog") increases the unilateral extensor muscle challenge (approximately 27% MVC in one side of the lumbar extensors and 45% MVC in the other side of the thoracic extensors) but also increases lumbar compression to well over 3000 N (Callaghan, Gunning, and McGill, 1998). This exercise can be enhanced with abdominal braCing and deliberate mental imaging of activation of each level of the local extensors. Once again, technique for challenging the extensors should be guided by the patient's status and goals. This is an individual clinical decision. Clinicians should remember to emphasize abdominal bracing and a neutral spine throughout all versions of this exercise. Poor form includes "hip hiking" or any other configuration that causes deviation (twist, flexion, or lateral bending) to the spine (see figure 13.8, a-b). Figure 13.8 Common mistakes made in the birddog are "hiking" the hip, which twists the spine (a) or not achieving a neutral spine-a flexed posture is seen in (b). 254 Low Back Disorders g, Remedial The starting position is on the hands and knees with the hands under the shoulders and the knees directly under the hips (a). For the patient with a very deconditioned back, this exercise involves simply lifting a hand or knee about an inch off the floor (b). After the patient is able to raise a hand or knee without pain, he can progress to

raising the opposite hand and knee simultaneously. g, Beginner's g, Intermediate The progression continues with raising one leg or one arm at a time. The intermediate birddog is achieved when the patient is able to raise the opposite arm and leg simultaneously. He should avoid raising either the arm or the leg past horizontal. The objective is to be able to hold the limbs parallel to the floor for about six to eight seconds. Good form includes a neutral spine and abdominal bracing. ��dQg/Advanced Developing the Exercise Program 255 To develop the motor control challenge further, the patient should not rest by placing the hand and knee to the floor after each holding repetition. Rather, after extending the normal birddog position (a), he should "sweep" the floor with the hand and knee so that no weight is borne by either.The patient is shown beginning the sweep (b), (c) shows him at the innermost point of the sweep,and (d) shows him coming back out of the sweep. The patient then extends the active limbs back out into the birddog position (e). This technique also allows muscles to reoxygenate with each "sweep" cycle (McGill, Hughson, and Parks, 2000). 256 Low Back Disorders Beginner's Program for Stabilization Now that you are familiar with some specific recommended low back exercises, how do you put these together to create the best possible program for your patient? We recommend considering the folloWing sequence: 1. Begin with the flexion/extension cycles, also called the cat/camel motion (see figure 13.9, a and b) to reduce spine viscosity. Note that the cat/camel is intended as a motion exercise, not a stretch, so the emphasis is on motion rather than "pushing" at the end ranges of flexion and extension. We have found that five or six cycles are often sufficient to reduce most viscous stresses-additional cycles rarely reduce viscous friction further. Those with sciatica may find increased symptoms during the flexion phase. Use pain to guide the suitable pain-free range of motion. 2. Slow lunges, with an upright and braced torso, satisfy the objectives of sparing the spine while achieving hip and knee endurance and mobility (see figure 13. 1). 3. Sciatics may try the nerve-flossing technique. 4. These motions are followed by anterior abdominal exercises, namely, appropriate curl-ups. 5. Lateral musculature exercises follow, namely, the side bridge for quadratus lumborum and the muscles of the abdominal wall for optimal stability. 6. The extensor program consists of leg extensions and the birddog. In general, we recommend that the isometric holds performed in the curl-ups, bridges, and birddogs be no longer than seven or eight seconds, given recent evidence from near infrared spectroscopy, indicating rapid loss of available oxygen in torso muscles contracting at these levels. ShOlt relaxation of the muscle restores oxygen (McGill, Hughson, and Parks, 2000). The endurance objectives are achieved by building up repetitions of the exertions rather than by increasing the duration of each hold. Motivated by the evidence for the superiority of extensor endurance over strength as a benchmark for good back health, we recently documented normal ratios of endurance times for the torso flexors relative to the extensors and lateral musculature (see chapter 11). Use these values to identify endurance deficits-both absolute values Figure 13.9 Cat/camel exercise. Note that this is a motion exercise and not a stretch; do not push at the end range of motion. Viscosity is measurably reduced after just a few cycles. Developing the Exercise Program 257 a nd for one muscle group relative to another-and to establish reasonable endurance goals for your patients. References Andersson, E.A., Oddsson, L., Grundstrom, O.M., Nilsson, J., and Thorstensson, A. (996) EMG activities of the quadratus l umborum and erector spinae muscles during flexion-relaxation and other motor tasks. Clin ical Biomechanics, 11: 392-400. Axler, c., and McGill, S.M. (997). Low back loads over a variety of abdominal exercises: Searching for the safest abdominal challenge. Medicine and Science in Spons and Exercise, 29 (6): 804-81 1. Biering-Sorensen, F. (984) Physical measurements as risk indicators for low-back trouble over a one-year period. Spine, 9: 1 06- 1 19. Butler, D.S.

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by causing exacerbating spine loads. We therefore suggest beginning exercises on a stable surface and establishing a positive slope to improvement. Introduce labile surfaces judiciously only once the patient has achieved spine stability and sufficiently restored load-bearing capacity, and can tolerate additional compression. This same principle can be extended to sitting. Sitting on a gym ball greatly elevates spine load through increased muscle coactivation. For this reason, nonpatients should avoid prolonged sitting on gym balls, and patients should use them only once they have achieved spine stability and increased loadbearing capacity. There is a time and a place for labile surfaces. Figure 14.1 The simple curl-up was assessed for the effect of a labile surface on muscle activation patterns. The percent of maximal voluntary contraction (% MVC) in the rectus abdominis and external and internal obliques caused by simply moving from a stable surface (a) to varying types of labile surfaces (b), (c), and (d) is shown in figure 14.2. 50 40 0 30 > ::2 -:R. 0 20 10 0 a b c (Postures shown in figure 14.1) D Rectus abdominis • Ext oblique D int oblique d Figure 14.2 The % MVC caused by each of the postures shown in figure 14.1. A curl-up with the body over a ball and the feet on the floor (figure 14.1 c) virtually doubles the abdominal muscle activation seen in a curl-up on a stable surface (figure 14.1 a) and, correspondingly, the spine load. Note that the % MVC required of the three muscles studied is also much higher in curl-ups with the body over a ball and the feet on a bench (figure 14.1 b) and with the body on a wobble board (figure 14.1 d) than on a stable surface. Clearly, a gym ball can be wonderful for advanced training but is contraindicated for many patients. Figure 14.3 The typical back extensor devices will impose generally twice the muscle loading of the birddog since both right and left sides of the lumbar and the thoracic extensors are activated. These may be necessary for some types of performance training, but they are ill-advised for most patients. Advanced Exercises 261 • Training with machines and equipment. Generally, the goal of establishing stabilizing motor patterns requires the individual to support body weight and coordinate the stabilization of all joints involved in the task. In other words, it is a whole-body, even wholeperson, endeavor. Many training machines, on the other hand, are made to isolate a specific joint. Since neither workers nor athletes perform their tasks in this artificially stabilized manner, these types of motor patterns may not be transferable and, worse, may cause inappropriate grooving of motor/motion patterns. The only time machines that isolate joints can be helpful is when an injury to a specific body part requires its protection during rehabilitative training. Some incorrectly believe that isolating a joint somehow reduces the loading and thus reduces the risk. Consider, for example, the typical extensor benches or Roman chair, which generally imposes twice the muscle loading than results from performing the birddog since both right and left sides of the lumbar and the thoracic extensors are activated (see figure 14.3), Patients who must use machines should consider certain backsparing techniques. The leg press rack, for example, sometimes causes the pelvis to rotate away from the back rest when the weight is lowered (see figure 14.4). The resultant lumbar flexion produces herniating conditions for the disc! If patients must use this exercise, we suggest they use just one leg at a time to ensure that the pelvis remains in contact with the back pad and that they preserve a neutral spine (see figure 14.5). Having stated thiS, however, we would never attempt to groove these motor patterns in any of our programs; they are nonfunctional except for leg-pushing a weight up a ramp! For the purposes of this discussion, cables to weight stacks are not considered machines since they do not isolate joint motion and add resistance to wholebody motion patterns. One particular exercise that enhances the ability to stabilize the spine is the cable pulldown. This 262 Low Back Disorders Figure 14.4 For back patients who have compromised hip flexion, lowering the weight sometimes causes the pelvis to rotate away from the back rest. This also

can occur if the weight is lowered too far. This produces herniating conditions for the disc! Figure 14.5 For patients who must use the leg press exercise, we suggest they use just one leg at a time to ensure that the pelvis remains in contact with the back pad and that they preserve a neutral spine. (Note that we still consider this a nonfunctional motor/motion pattern). Figure 14.6 Two variations, (a) and (b), of the cable pulldown exercise should be performed with the handlebar lowered to the chest rather than to the back. This enhances the role of several spine extensors and in particular the latissimus dorsi for lumbar stabilization. exercise, when the handlebar is brought down to the chest (rather than the back, which is traditional technique) challenges the latissimus dorsi and other stabilizers (see figure 14.6, a-b). As we will see in a subsequent section, these types of exercises form a part of a performance-based program. Advanced Exercises 263 Safely Progressing Back Exercises The curl-ups, side bridges, and birddogs pictured in chapter 13 can be made more challenging and yet still incorporate the safety features noted. After explaining these advanced forms of the big three exercises, I will present an additional advanced back exercise. p, Highest Level Once the patient has mastered the advanced curl-up described in chapter 13, she may be instructed to try the following: 1. Brace the abdomen (if necessary, review the instructions on pages 210-213). 2. Curl up against the brace, but do not curl up any higher than in other forms of the exercise. 3. Perform deep breathing while in the "up" curl-up position and while maintaining bracing. Remain in the up position long enough to take a few deep breaths. This level of curl-up will be challenging for the toughest NFL linebacker! ge, Highest Level The side bridge can be more challenging by incorporating the roll described in chapter 13 (page 252), together with dynamically contracting and relaxing the abdominal wall while sustaining position. g, Highest Level The birddog is more demanding when the patient consciously stretches the hand out forward and the foot out behind-all the while ensuring that spine motion (particularly bending) does not take place. If performance objectives are desirable and if the patient has no exacerbation of symptoms, small wrist and ankle weights may be allowed. When searching for ways to obtain maximum myoelectric activity from the back extensor muscles, we discovered that isometric back extensor exercises do not recruit the full pool of motor units. Many more motor units fire with some extensor motion. As a result of this discovery, this back extensor exercise, which builds strength and endurance and helps to hypertrophy the muscles, was developed. While the athlete is lying prone on a bench with the torso supported on a movable stiff pad, place weight in one hand.The edge of the pad is placed under the mid ribcage. The cantilevered portion of the spine is slightly flexed and then extended, combined (continued) 264 Low Back Disorders Advanced Exercises for Back Strength and Endurance (continued) with some slight twist back to neutral (a). The object is not to actually twist but to focus activation on one side of the extensors-to use mental imagery (chapter 11) to assist in activating the maximum number of available motor units. The spine never extends past neutral. After a set, place the weight in the athlete's other hand and have the athlete repeat the exercise. Then move the pad downward (perhaps about the level of the navel) so that a greater portion of the torso is cantilevered (b). Have the athlete repeat the sets. Move the pad farther down the athlete's body (perhaps to about the pelvis), leaving more of the torso cantilevered, and have the athlete repeat the entire process (c). Another variation of this exercise is to perform the extension motion over a gym ball where the feet are secured (d) and the ball is moved progressively toward the pelvis with each set (e). (See Goldenberg and Twist, 2002) or the legs are cantilevered (f) and the ball moved progressively toward the chest (g). While some strength athletes have successfully used this exercise to enhance performance, we are still quantifying the muscle activation and joint loads. Full data will be presented on this and

other "strength" exercises in my upcoming book for athletic performance. ---- Advanced Exercises 265 Occupational and Athletic Work Hardening Certain workers and athletes encounter demands that require training that we would never recommend to patients unless they were absolutely necessary. Anyone considering going beyond the exercises described previously should be fully aware of the increased risks of back injury that these exercises will pose. These exercises aimed at workers or occupational athletes should be restricted to that group because they present a level of risk that is unacceptable for any but those who are willing to assume them for the sake of athletic or work performance. Once again, chOOSing the appropriate challenge involves a blend of clinical art and science. Too little load will not produce the training adaptation, and too much will break tissue down. Listen to the body, and give adequate rest. Low Back Exercises for High-Performance Workers or Athletes I will be writing a book in another year or so on evidence-based high-performance training for the back. Given the emphaSis of this book on disorders, this section is brief and introductory. In general, we stage training of the athlete with the same steps used in our rehabilitation approach, only two more steps are added. We begin in stage 1 by identifying the essential motions and grooving appropriate motion/motor patterns. Stage 2 is directed toward ensuring joint and whole-body stabiliZing patterns. Stage 3 is to develop muscle endurance around these patterns. Stage 4 is directed to enhanCing strength. And, finally, stage 5 is to establish power. Unfortunately, too many exceptional athletes are trained, or given a rehabilitation program based on strength and power, without an adequate foundation of stabiliZing motion/motor patterns. They end up with back problems and are referred to us. In this section we will briefly consider exercises that may be helpful to either workers with demanding jobs or athletes. The requirements of the activities are not excessive for those who have adequately prepared by mastering all of the exercises previously discussed in the text. Specifically, these individuals should have mastered spine position awareness and be able to produce low back stabiliZing patterns but may need more work to successfully move through the ranges of motion specific to their task. In addition, they may need more strength and endurance. 266 Low Back Disorders Qualifying the Worker/Athlete The clinical decisions involved in staging a worker/athlete through the progression of challenge is an art that can be assisted with scientific data. Those who are looking for a set recipe, however, will fail at creating a successful program for an individual. Generally we blend the worker/athlete's current exercise status and history of injury with our own knowledge of spine tissue loads that result from various activities and our knowledge of injury mechanisms to qualify an individual for a specific exercise progression. We then put our educated guess to the test and monitor patient progress to ensure the maintenance of a positive slope of improvement in symptoms and function. Lumbar Stability With Elevated Simultaneous Physiological Work Rates W hile some individuals can maintain spine stability over all sorts of activities, including those that require the stabilizing musculature to assist with physiological challenges such as elevated breathing, others cannot (McGill, Sharratt, and Seguin, 1995). We have noted a compromise in the ability to stabilize more often postinjury (McGill et aI., in press), although we have also detected that compromise in "virgin backs." Interestingly, tall athletes tend to be poorer in their ability to cocontract the abdominals to ensure sufficient stability during high work rates and highly challenged breathing than their shorter counterparts. This perception appears to be supported by the most recent evidence showing that taller workers have a greater likelihood of having perturbed motor patterns while breathing heavily and holding spine loads (McGill et aI., in press). Consider the warehouse worker, firefighter, or football player who must work at a high physiological rate that results in deep and elevated lung ventilation. The inability to maintain constant

cocontraction in the abdominal wall (i.e., the muscles tend to relax during deep inhalation) is an indicator of compromised spine stability, partiCLIlady when heavy external loads that demand a stable spine are required. These individuals must develop stabilizing motor patterns that will transfer to all activities. ablishing Spine Stability When Breathing Is Elevated One training exercise we have developed is to have the individual 1. ride an exercise bike at an intensity to elevate ventilation, and then 2. immediately dismount and adopt the side-bridge posture on the floor. In this position the stabilizing musculature must remain isometrically contracted; otherwise, the bridge posture is lost. Heavy ventilation is also required, however, acting to groove the motor patterns that coordinate diaphragm contraction and other thoracic muscles involved in efficient lung function. The curl-up and birddog postures are also used after vigorous stationary biking to groove stable patterning in all of the supporting muscles. In this way we establish spine stabilization patterns in workers and athletes alike. Torsional Capability Some activities that athletes or workers must perform require twisting and the creation of substantial torsional moments. The difference between the mechanics involved in twisting and the generation of torsional moments was discussed in chapter 5. The question is, how can we maximize stability and minimize the risk of injury during Advanced Exercises 267 training for trunk torsion? Because, as previously noted, generating torque about the twist axis imposes approximately four times the compression on the spine as an equal torque about the flexion/extension axis, it is unwise to train for torsion generation until the back is quite healthy. g for Torsional Capacity The technique we have found for producing low spine loads while challenging the torsional moment generators is to raise a handheld weight while supporting the upper body with the other arm and abdominally bracing (see a and b) to resist the torsional torque with an isometrically contracted and neutral spine. Dynamic challenged twisting is reserved for the most robust of athletes. We would never recommend training on the torsional machines (such as those where the athlete is seated with the pelvis belted down and the upper body twists against resistance) unless specific athletic performances were the training objective,and then the individual must be made aware of the elevated risk of pursuing this approach. Some very useful and transferable motion patterns are produced with the various cable exercises (e.g., high pulls, low pulls, and "chopping" exercises), which produce high torsional challenges. Because of the lumbar compressive loads associated with these torsional torque challenges, we suggest discipline in bracing the torso in a neutral posture. It would also be wise to consider the transmissible vector (the perpendicular distance between the cable force and the lumbar spine) when choosing the technique to control the torsional torque and resultant spine load. Antalgic Posture From Sitting At our university clinic we notice a rash of back troubles at exam time. The students are sitting too long studying. Typically, they sit for a long spell and then when they stand they have the typical antalgic posture, leaning forward at the hips and unable to immediately stand upright. 268 Low Back Disorders Special Suggestion for Those Whose Back Is Worse With Sitting , and Then Have Trouble Standing For these patients I may suggest 1. putting on a backpack weighing about 5-10 kg (11-22 lb) and 2. going for a walk over rough ground for an hour or two. Amazingly (to some), many patients report that this works wonders.The typical forwardflexed antalgic posture requires the back extensors to be active (which imposes a substantial load penalty on a flexed spine).The backpack acts as a counterweight to extend the spine and bring the torso upright. The back extensors are no longer needed to contract, effectively removing their contribution to spine load. Wearing the backpack reduced spine loading! Low Back Exercises Only for Athletes Specific athletic objectives require specific training techniques. As noted earlier, however, too many patients make the mistake of looking to athletes for training exercises under the misconception that

the same approach will help their own backs. This critically important notion must be emphasized again: Athletic exercises are not for enhancing back health in patients. In addition, too many athletes use bodybuilding principles for building mass. These must not be emulated by patients. The following exercises are reserved for athletes only.
Training-Specific Athletic Maneuvers The various trunk torsional machines or resisted twisting motions are reserved for those who want to excel at velY special tasks such as Olympic discus throwing. (I must add that some track athletes with whom I have been associated clinically do not perform these exercises because it exacerbates their back symptoms.) Othelwise, the torsional moment capabilities are developed with the spine in a neutral position, with a fully braced torso musculature, but with a torsional moment challenge. The neutral position is the most robust posture to withstand the elevated spine loads; it is also the spine posture that is transferable to other activities that require torsional moments with the least risk of spine damage. Sprinting and other power running events such those performed by football players should be addressed here. Top-end speed for most sprinters is usually limited by the recovelY of the leg in flexion Chip flexion), not a lack of hip extension power. Thus, many train the psoas according to the power philosophy-and end up with a bad back. It is critical for these athletes to maintain spine stability and be conscious of the braced and neutral spine during rigorous hip flexor training required for sprinting events.
All sorts of exercises and special equipment have been developed for performance training. But very few performance approaches have been documented with substantial scientific foundation to warrant discussion in a book such as this. However, one exception is the book Supertraining by Dr. Mel Siff, where an impressive number of studies have been synthesized and reviewed. For example, Dr. Siff makes a case for avoiding the development of motions that are guided predominantly by machines, and instead advocates cables and free weights with data-based argument. Further, substantiated principles of functional anatomy, physiology, biomechanics, and motor aspects such as facilitation are used to justify the approaches documented in Supertraining to enhance strength, power, speed, and muscle endurance. These performance objectives cannot exist without the foundation of stability, mobility, and the additional principles I have documented in this book. While there is no question that these principles and exercises are excellent Advanced Exercises 269 for performance, there is a high resultant spine load. Some backs simply will not tolerate some of these higher-demand exercises-but not everyone can tolerate the rigors of training to be a champion, regardless of the soundness of the scientific technique. The exercise variations are endless and are outside of the scope of this book. Finally, when designing workouts, the "big three" can be performed at the beginning of the workout to help groove and establish the stable motor patterns for the rest of the training session. Some trainers also like to finish the session with these exercises if they desire to take the exertions to fatigue. One method growing in popularity is known as the star exercises, which are designed to promote stability throughout a range of motion. A plus sign (+) is drawn on the floor. The patient begins with the feet on the center of the + and performs various practice movement patterns, moving into each quadrant. What is performed in one quadrant is repeated in another quadrant for motion/motor symmetry. Various pairs of positions may be combined with different postures and may be done with or without resistance, which may be provided by either dumbbells or hand grips on cables. See, for example, the pairs (a) and (b),and (c) and (d). Star exercises are ideal for grooving patterns such as a tennis serve. It is crucial that you emphasize abdominal cocontraction and limiting lumbar motion to the patient's safety zone. Training to Squat and Power Clean Squats and power cleans are good exercises for developing power for athletic pelformance. The instructions from our perspective are velY simple: Never

sacrifice fonTI for lifting more weight. Year after year I give this advice to young athletes, and year after year a substantial proportion will ruin their backs by not heeding this simple guideline. As the old saying goes, "It's amazing how much your parents learned as you grew older." Very few people in North America can perform squats and power cleans well and with a low risk of injury. The Eastern Europeans are technical masters, and it is beyond the scope of this book to delve into the nuances of the technique. But it is interesting to see the emphasis placed on grooving the correct motion/motor patterns long before substantial weight is attempted. Young Eastern European athletes spend years developing the form by lifting broomsticks. Only when the form is perfect is strength increased and weight added to the bar. Generally, preserving the neutral 270 Low Back Disorders lumbar spine will solve many of the safety issues, but this depends on the ability to take the shoulders and hips to extremes in the range of motion. Some have found that placing the weight bar on blocks to raise the starting height improves the utility of this exercise for athletes o ther than competitive weightlifters. This way a lifter can accomplish a fast lift without the larger back loads associated with the initial crouched posture needed to pull from the floor. In this power exercise speed is important; participants are advised to "train slow to be slow, train fast to be fast." For those interested in these exercises, the actual weight kinematics for the full power clean and the coordination of the leg muscles, torso muscles, and those involved in the shoulder pull can be found elsewhere. Power athletes who are not weightlifters are given power lifting exercises with things like medicine balls (see figure 14.7, a-c) in our programs. We rarely recommend power cleans with a bar despite their Widespread use. Figure 14.7 Medicine ball lift (a) and (b) and toss (c) for power. Looking Forward Rehabilitation endeavors are continuing to embrace techniques that consider notions of torso stability and various components involving posture, motor patterning, and appropriate progressive challenge. Many groups continue to work • to understand the contributions to stability of various components of the anatomy at particular jOints-and the ideal ways to enhance their contribution; • to understand what magnitudes and patterns of muscle activation are required to achieve sufficient stability while sparing the joints; Advanced Exercises 271 • to identify the best methods to reeducate faulty motor control systems to both achieve sufficient stability and reduce the risk of inappropriate motor patterns occurring in the future; and • to develop motor patterns for optimal performance in athletes. Collegial efforts between scientists and clinicians continue to develop the scientific foundation to justify better low back injury prevention and rehabilitation approaches. Much remains to be done. References Goldenberg, 1., and Twist, P. (2002) Strength ball training. Champaign, 1L: Human Kinetics. McGill, S.M., Sharratt, M.T., and Seguin,].P. (1995) Loads on spinal tissues during simultaneous lifting and ventilatory challenge. Ergonomics, 38: 1772-1792. McGill, S.M., Grenier, S., P reuss, R., and Brown, S. (in press) Motor performance characteristics of the low back: Links to having a histOlY of low back troubles. Vera-Garcia, F.]., Grenier, S.G., and McGill, S.M. (2000) Abdominal response during curl-ups on both stable and labile surfaces. Physical Therapy, 80 (6): 564-569. THIS PAGE INTENTIONALLY LEFT BLANK Epilogue Now you have another perspective to employ in your efforts to prevent and rehabilitate bad backs. I hope that from now on, when you read that bad backs just happen, or that they cannot be diagnosed to guide therapy, or that they are solely the function of psychological factors or the compensation system, you will pause and reflect that you are reading the musings of individuals who have reached the end of their expertise. Not that anyone has all the answers. With each experiment that we perform we may obtain some new insight, but generally we are confronted with many more questions. So, with each experiment we become relatively more ignorant. And after hundreds of experiments we are so ignorant that

we are tempted to think that we are no longer qualified to make recommendations about the low back to anyone! Consider the viewpoint described in this text and blend it with your own clinical wisdom and experiences. The very best clinicians, with whom I have had the pleasure of working, had wonderful clinical skills and insights but made use of a scientific foundation. To the clinicians-I wish the confidence to continue with what you know works and the inspiration and leadership to try new approaches when things are problematic. To the students of clinical disciplines and those involved in the continuing development of the scientific foundation-there are so many more exciting experiments to perform on the wonderful fascination that we share, the low back. May we all enjoy the continuing journey. 273 Appendix A.l Raw Cross-Sectional Areas (mm2) (Standard Deviation in Parentheses) Measured Directly From MRI Scans Muscles Vertebral level - LS L4 L3 L2 R rectus abdominis 787(250) 750(207) 670(133) 712(239) --L rectus abdominis 802(247) 746(181) 693(177) 748(240) R external oblique 915(199) 1276(171) 1158(222) --L external oblique 992(278) 1335(213) 1351 (282) --R internal oblique 903(83) 1515(317) 1055(173) L internal oblique 900(115) 1424(310) 1027(342) R trans. abdominis 119(22) 237(82) 356(110) 596(50) L trans. abdominis 175(57) 224(48) 376(115) 646(183) R abdominal wall* 11 04(393) 2412(418) 3269(422) 3051 (463) L abdominal wall* 1146(377) 2420(475) 3329(468) 3111(556) R longissimus thor. 747(162) 1175(370) L longissimus thor. 782(129) 1089(251) R iliocostalis lumb. 1368(341) 1104(181) L iliocostalis lumb. 1395(223) 1150(198) R multifidus 447(271) 343(178) L multifidus 472(269) 366(157) R latissimus dorsi 232(192) 429(202) L latissimus dorsi 256(217) 372(161) R erector mass** 905(331) 2151(539) 2831 (458) 2854(547) L erector mass** 986(338) 2234(476) 2933(382) 2833(456) R psoas 1606(198) 1861(347) 1594(369) 1177(285) --L psoas 1590(244) 1820(272) 1593(291) 1211(298) R quadratus lumb. 725(209) 701 (212) 552(192) L quadratus lumb. 625(249) 746(167) 614(189) Disc area 1360(276) 1459(270) 1415(249) 1332(294) Total area 52912(9123) 51813(9845) 54286(8702) 55834(8112) *Abdominal wall includes external and internal oblique and transverse abdominis. ** Erector mass includes longissimus thoracis, iliocostalis lumborum, and multifidus. 2 74 --- Ll T1 2 - 576(151) 514(99) 1248(228) 1095(222) 1180(184) 1258(347) 1181(316) 921 (339) 1158(247) 835(400) 290(96) 289(66) 324(95) 312(76) 717(260) 1014(264) 682(260) 960(310) 2615(405) 2614(584) 2723(428) 2601 (559) 513(329) 330(210) 488(250) 462(190) 392(249) 320(197) 404(220) 326(5) 1334(285) 1241(166) 59091 (6899) 63287(9153) Vertebral level Tl l Tl O T9 T8 T7 T6 TS 938(49) 938(21) 556(234) 551 (1 70) 331(89) 351(90) 312(97) 327(80) 353(53) 355(73) 1254(281) 1368(330) 1458(269) 1581(159) 1764(289) 1876(432) 2477(246) 1102(316) 1239(257) 1417(293) 1582(281) 1697(189) 2013(422) 2596(721) 1832(282) 1690(210) 1413(304) 1049(201) 842(165) 777(189) 743(70) 2041 (285) 1722(279) 1471(351) 1129(100) 879(114) 779(95) 675(76) 1133(124) 1015(125) 933(112) 798(91) 797(104) 741(80) 671(82) 59249(7272) 61051(7570) 61732(6960) 65794(5254) 67782(3982) 66410(2372) 69337(2233) 275 A.2 Raw Lateral Distances (mm) Between Muscle Centroids and Intervertebral Disc Centroid (Standard Deviation in Parentheses) Muscles Vertebral level LS L4 L3 L2 R rectus abdominis 32(5) 38(7) 43(7) 46(8) L rectus abdominis -33(6) -36(7) -38(8) -43(7) R external oblique 125(13) 130(10) 140(5) L external oblique -120(9) -125(9) -133(7) R internal oblique 109(11) 116(8) 123(9) L internal oblique -103(9) -112(8) -121(11) R trans. abdominis 99(1) 108(11) 122(9) L trans. abdominis -101 (1) -101(9) -107(7) R abdominal wall* 102(8) 113(12) 119(8) L abdominal wall* -102(9) -115(14) -114(7) R longissimus thor. 22(4) L longissimus thor. -19(5) R iliocostalis lumb. 52(4) L iliocostalis lumb. -48(6) R multifidus 11 (1) L multifidus -14(7) R latissimus dorsi 102(8) L latissimus dorsi -104(15) R erector mass** 22(6) 34(7) 40(4) L erector mass** -21(5) -33(6) -38(5) R psoas 54(4) 50(3)

44(3) L psoas -54(4) -48(4) -42(3) R quadratus lumb. 81 (5) 75(6) L quadratus lumb. -78(12) -73(4) Total area 0(2) 1 (3) -2(4) *Abdominal wall includes external and internal oblique and transverse abdominis. ** Erector mass includes longissimus thoracis, iliocostalis lumborum, and multifidus. 2 76 117(9) -109(9) 123(9) -120(9) 32(2) -30(6) 58(4) -60(10) 13(4) -12(3) 108(8) -107(9) 42(4) -41(6) 39(2) -38(3) 63(5) -64(5) -1(3) L1 T12 37(8) -35(17) 32(6) 30(2) -37(12) -34(4) 68(10) 65(7) -65(9) -67(7) 13(3) 10(3) -11(3) -11(2) 122(12) 129(10) -117(11) -128(7) 44(5) 42(3) -41(7) -40(4) 32(3) 32(3) -31(3) -32(2) 46(6) 46(11) -50(6) -47(5) -1(4) 0(3) Vertebral level T11 T10 T9 T8 T7 T6 TS 29(1) -36(7) 61(4) -67(11) 8(2) 11(2) 12(2) -12(2) -12(2) -15(10) 129(9) 140(9) 141(8) 145(7) 146(7) 153(7) 153(4) -129(10) -137(9) -139(8) -143(6) -147(10) -153(5) -151 (5) 34(4) 34(4) 32(4) 31 (7) 30(4) 25(5) 27(2) -40(3) -36(3) -35(4) -33(6) -31(2) -29(3) -27(6) 1(3) 0(2) 0(2) 2(1) 2(1) 1(3) 2(3) 277 A.3 Raw Anterior/Posterior Distances (mm) Between Muscle Centroids and Intervertebral Disc Centroid (Standard Deviation in Parentheses) --Muscles Vertebral level LS L4 L3 R rectus abdominis 81 (16) 73(14) 79(13) L rectus abdominis 80(15) 73(14) 81(14) R external oblique 35(10) 20(14) L external oblique 32(18) 19(11) R internal oblique 41 (12) 25(9) L internal oblique 41(17) 26(12) R trans.abdominis 55(0) 28(11) 22(11) L trans.abdominis 50(5) 30(14) 23(10) R abdominal wall* 58(16) 31 (12) 17(12) L abdominal wall* 59(17) 32(13) 20(12) R longissimus thor. -61(6) L longissimus thor. -61(5) R iliocostalis lumb. -57(7) L iliocostalis lumb. -57(7) R multifidus -55(7) L multifidus -53(7) R latissimus dorsi -45(16) L latissimus dorsi -43(17) R erector mass** -64(6) -61(5) -61(5) L erector mass** -63(5) -61(5) -61(5) R psoas 18(9) 1 (5) -7(5) L psoas 19(8) 2(4) -6(4) R quadratus lumb. -36(9) -37(6) L quadratus lumb. -31(5) -34(6) Total area 1 (1 0) -2(9) 1 (8) *Abdominal wall includes external and internal oblique and transverse abdominis. ** Erector mass includes longissimus thoracis, iliocostalis lumborum, and multifidus. 2 78 L2 91(14) 92(14) 28(12) 28(11) 36(17) 40(16) 36(6) 44(5) 30(15) -31 (11) -62(7) -63(6) -61(7) -61(6) -55(6) -56(5) -47(12) -46(10) -61(5) -62(5) -9(5) -8(2) -37(6) -36(5) 9(8) Ll T T;; --- 109(8) -112(6) -60(7) ---60(7) . -62(5) -- - [illegible] -60(6) - -59(8) -- --59(7) -61(4) -58(6) --52(6) -51(3) - -51(5) -50(3) - -47(10) -39(8) -- -46(7) -37(8) -59(5) -56(5) -60(4) -57(5) -11 (6) -14(2) -11(4) I -11 (1) -3[illegible] ----31 (6) -34(4) -32(8) 18(5)-1 -24(7) -- -- - ---------- Vertebral level ---- -- -- T1 1 T1 0 T9 T8 T7 T6 T5 1-- --- ---56(4) -52(4) -57(1) -56(2) -- f-- --- -- -47(5) -49(4) -48(2) -- -47(5) -47(3) -47(2) ---32(7) -24(7) -22(7) -18(9) -17(6) -12(3) -17(5) -28(9) -23(7) -19(7) -17(7) -15(8) -11 (7) -19(3) ---54(4) -54(4) -52(4) -52(3) -52(4) -47(4) -50(3) -52(4) -52(4) -51(4) -51 (3) -51(4) -46(5) -50(3) - 29(8) 30(9) 32(7) 36(7) 37(6) 32(10) 34(5) 279 280 Photo Credits Steve Brooks: Figure 9.4 Stuart McGill: Figures 1.3a; 2.2, a and b; 2.3; 4.7; 4.9; 4.12; 4.13; 4.14, a and b; 4.17; 4.21; 4.22; 4.24; 4.25; 4.26, a through d; 4.32, a through d; 4.37; 4.38; 5.12; 5.13, a and b; 5.14; 5.15; 6.1, a and b; 6.5, a and b; 8.2; 8.3, a and b; 8.6, a and b; 8.7; 8.10, a and b; 8.11; 8.13, a and b; 8.14; 8.15, a through f; 8.17; 8.18; 8.19, a and b; 8.20, a and b; 8.24; 8.25, a and b; 10.1, a through c; 11.2, a and b; 11.3a; 11.4, a through d; photo on page 233; figures 13.3a; 13.5; 14.1, a through d; photos on page 264; photos on page 265; figure 14.7, a through c. William Marras: Figure 7.1 Glossary anisotropic-Behavior of an anisotropic material is dependent on the direction of the applied load. For example, adult bone is stronger in compression than in tension. antagonist-A muscle that opposes the function of another in terms of torque generation but that may be necessalY to ensure a stable joint. breaking strain-Strain at which tissue fails. buckle-The spine may experience several modes of unstable behavior. The column may buckle under compressive loads if sufficient stiffness is not present in the rotational degrees of freedom. The buckle is abnormally large rotation at a single joint, resulting in very large passive tissue forces. dermatome-An area of skin supplied with afferent nerve fibers by single spinal nerve root. eigenvalue-The element of the

Hessian matrix that, if less than zero, indicates the potential for possible unstable behavior. eigenvector-Describes the mode and site of unstable behavior, and what deficit "allowed" the instability to develop. electromyography-The measurement of the electromyogram, or electrical event associated with muscle contraction. etiology-Analysis and description of the cause of injury or disease. failure tolerance-Load at which a tissue fails. lordosis---The curvature of the lumbar spine where neutral lordosis is the natural curve of the spine, hypolordosis occurs with a flexed lumbar spine, and hyperlordosis occurs with an extended lumbar spine. moment-Moment of force; occurs when a force is applied through a distance about an axis to create rotational effort. moment arm-The perpendicular distance between the axis of rotation and a force, which creates a moment. myoelectric-Of or relating to an electric signal obtained from contracting muscle. myotome-Electric signal obtained from contracting muscle. neutral lordosis---When the lumbar spine is neither flexed nor extended. NIOSH-National Institute for Occupational Safety and Health. nociception--Sensation of pain with a nociceptor (pain-sensing organ). odds ratio--The increase in odds of an event occurring. (Full explanation via an example is provided on page 30.) osteophytes---Bony spurs that grow on the stress points of a vertebra usually indicative of an unstable joint. 281 282 Glossary palpation-Using the hands to feel for spinal motion or pathology. pars interarticularis-Part of the vertebra that connects the facet articulating surface to the neural arch and is the site of fracture. pathomechanics-Mechanics associated with pathology. Poisson's effect-A material property of an elastic sheet in which strain longitudinally produces proportional strain in the lateral axis. provocative/functional diagnosis-Diagnosis based on tasks that exacerbate symptoms. provocative testing-Provoking tissues with movement or direct load to elicit pain. psychophysical--Variables that are associated with psychological or social aspects of an individual. psychosocial-These variables influence behavior and include psychological traits such as perception of control over a task and social traits such as how individuals are viewed by others. spondylolisthesis-A fracture of the pars that causes an anterior shear displacement of the superior vertebrae (usually L4 or L5). strain-A change in length normalized to rest length (dimensionless ratio) and expressed as a percentage. subfailure-An applied load that is less than the load required to cause damage if applied once. synergist-A muscle that assists another in the same functional objective. tolerance-A load value above which injulY occurs. It is modulated by repetition, duration, rest, and so on. transmissible vector-The transmissible property of any force vector is due to it having an effect along the line on which it is directed. A perpendicular line can be drawn from the force vector to any body joint to determine the moment arm, or the potential of that force to cause a moment. viscoelastic-The property of being both elastic and viscous that absorbs the shock of loads. viscosity - Friction as a function of velocity; it always moves in the direction of motion or impending motion. yield strength-The load at which a tissue begins to experience damage. Index Nole: Tables are indicated by an italicized t following the page number; figures by an italicized.! A Aaras, A. 162 abdominal belts about 194 for athletes 191 biomechanical studies 196-198 clinical trials 195-196 'prescription guidelines]99-200 psychophysical studies 1 99 and range-of-motion restriction 197-198 studies of heart rate and blood pressure 198-199 for workers 178 abdominal bracing 210-21 1 , 212f, 213, 248, 253 abdominal contraction, maintaining 210-211, 213 abdominal fascia 68, 69f, 8V abdominal hollowing 2 10-21 1, 2qf, 213 abdominal muscles about 68-71 activating specific portions of 7 / activation with flexion tasks 10V exercises 72 abdominal wall about 70-71 cocontracting independent of lung ventilation patterns 220 as lumbar torso stabilizer 244 during sitting 1 04 accessOIY process 46f, 51 activity intolerance, and pain 8 Adams, M.A. 55, 1 1 3, 1 14, 123-1 24, 125, 198 adipose tissue pads 52/ aerobic exercise, in exercise

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